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PREANESTHETIC ASSESSMENT

Elizabeth A. M. Frost, *Guest Editor*

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Preanesthetic Assessment of the Oncology Patient 867
Richard B. Patt

Advanced cancer is a multisystemic disorder characterized by obvious or occult alterations in physiologic function that has important implications to the patient's response to anesthesia and surgery. In addition to abnormalities related to direct invasion, local pressure, and humoral mechanisms, patients are exposed to antitumor therapy and its sequelae. Finally, patients are multisymptomatic. Ongoing pain treatment and other palliative care also must be considered when planning anesthetic management.

Preanesthetic Assessment of the Patient with Acquired Immunodeficiency Syndrome 881

Alexander W. Gotta

Preoperative assessment and anesthetic management of the patient with acquired immunodeficiency syndrome challenges the anesthesiologist to deal successfully with a wide variety of clinical syndromes representing widespread infection and damage in various organ systems. Disease of the central and peripheral nervous systems may contraindicate the use of regional anesthesia. The airway may be compromised by lymphoid hyperplasia and infection. Direct infection of the heart with human immunodeficiency virus causes serious cardiac arrhythmias and congestive heart failure. Diarrhea and malnutrition create electrolyte and fluid imbalances.

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FORTHCOMING ISSUES

March 1991

FIBEROPTICS IN ANESTHESIA

James T. Roberts, MD, *Guest Editor*

The use of fiberoptic endoscopic equipment in anesthesia is so important and essential to the administration of high quality anesthesia and intensive care that all anesthesiologists should have the equipment immediately available in their practice. An anesthesiologist may need to use a fiberoptic endoscopic piece of equipment either emergently or electively, with single lumen or double lumen tubes, or for diagnostic or therapeutic reasons. This issue of *Anesthesiology Clinics of North America* on Fiberoptics in Anesthesia is an excellent treatment of this new, rapidly changing and now essential aspect of good anesthesia and intensive care. This excellent treatment results from the guest editor, Dr. James Roberts, who is extremely well qualified in this field and the stellar group of authors he has assembled to write about the subject.

RECENT ISSUES

September 1990

HEMORRHAGIC DISORDERS

Bruce D. Spiess, MD, *Guest Editor*

June 1990

POSTANESTHETIC CARE UNIT PROBLEMS

Gerald Shapiro, MD, MPA, *Guest Editor*

March 1990

OBSTETRIC ANESTHESIA

Laurence S. Reisner, MD, *Guest Editor*

Foreword

Proper anesthesia care must begin, by definition, with an appropriate preanesthetic assessment of the patient. Potential problems must be recognized and anticipated (by preanesthetic) and therapy eliminated or minimized; this is the only way that the risk of perianesthetic complications can be reduced greatly. Thus, the beginning as well as the cornerstone of good anesthesia is proper preanesthetic assessment. All major organ systems and the special problems posed by all subspecialty surgery are within the province and concern of the anesthesiologist during the preanesthetic assessment. This issue of *Anesthesiology Clinics of North America* on Preanesthetic Assessment completely covers how to uncover the important organ- and surgery-related preanesthetic problems.

Anesthesiology Clinics of North America is fortunate to have Dr. Elizabeth Frost as the guest editor for this important and broad-based topic; she is a natural and logical best choice for several reasons. First, her own training and clinical experience and expertise is extremely broad based. She has had postgraduate training in surgery, medicine, obstetrics, general practice, and of course, anesthesiology. Second, she is an acknowledged expert in several anesthesia subspecialties and is presently the Director of Neuroanesthesia in a major-league hospital. Third, Dr. Frost has had extensive editorial experience. Presently, she is on the editorial board of *Anesthesiology News, Practical Reviews in Anesthesiology,* and *The International Trauma and Critical Care Society Newsletter* and is a prominent leader of several history of anesthesia programs. She has singly edited three books on anesthesia for neurosurgery (one being a previous issue of *Anesthesiology Clinics of North America)* and other books on postanesthetic care unit practice and preanesthetic assessment. Of most relevance to this issue has been a simply outstanding series of lessons. Fourth, her many published papers, abstracts, and chapters in books span all of these previously mentioned areas, and her writings additionally reveal quality involvement in respiratory therapy, monitoring, intravenous and inhalational anesthesia, management of airway problems, acupuncture and postoperative pain relief, outpatient anesthesia, and the feminist movement in anesthesiology. In conclusion, the great intrinsic clinical and educational value of the subject of Preanesthetic Assessment is greatly enhanced by the guest editorship of Dr. Elizabeth Frost.

Jonathan L. Benumof, MD
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Preface

Dr. John Snow noted prior to 1850 that respiratory and cardiac diseases, pregnancy, alcoholism, and age could all modify the effects of inhalational agents. He wrote, "It is desirable to pay attention to every circumstance connected with the health and constitution of the patient before exhibiting chloroform as many of these circumstances influence its effects." He also commented on the importance of a physical examination prior to anesthesia as a means of detecting abnormalities (especially cardiac dysrhythmias) that might later be attributed erroneously to adverse effects of the anesthetizing drug.

But little attention was paid to his advice on preanesthetic assessment. Indeed, at the beginning of this century, anesthesia was often administered by anyone in the operating room, whether trained in anesthetics or not. The premise was that surgery must be performed, and anesthesia reduced the pain and horror of the knife. Many patients died—it was the will of God.

Advances in anesthesia and hence in surgical capability in the last 30 years have been enormous. As patients age, they develop more and varied diseases. Preanesthetic assessment and appropriate corrective therapy became crucial to good outcome.

I became interested in collecting articles on preanesthetic assessment of different patient groups and disease states first as a continuing medical education series and as a means to help junior staff members organize their thoughts on paper. Later, I realized how important accurate evaluation of pathologic conditions and drug ingestion can be to anesthetic management.

In this issue, I have attempted to cover most of the major facets of preanesthetic care. Again, in the main part, the contributors are more junior members, principally from my institution, who have developed particular interests in different areas.

The issue is intended for residents and general practitioners in both anesthesia and surgery. It is a clinical text and makes no pretense at extensive literature or laboratory review.

Although it is clear that practice varies with locale throughout the United States, it is hoped that all readers will find some common ground and be able to apply the principles expressed in the preanesthetic evaluation of at least some of their patients.

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Preanesthetic Assessment of the Patient with Cardiac Disease

*Sharad Khetarpal, MB, BS, FFARCSI**

More than one million patients with heart disease undergo cardiac and noncardiac surgery annually.[17] Perioperative morbidity and mortality rates are increased in this population group.[6, 26] Preanesthetic assessment involves the identification of predictors of anesthesia and surgery outcome and recognition and diagnosis of cardiac disorders, so that an appropriate anesthetic course can be planned.

PREOPERATIVE PREDICTION OF CARDIAC MORTALITY AND MORBIDITY

Previous Myocardial Infarction

The risk of perioperative myocardial infarction (MI) in the general population is low (<0.2%),[7] whereas the risk in patients who have had a previous MI is approximately 6%. In those who have had coronary revascularization after infarction the risk again becomes low 1 month after the procedure.[7] Surgery within 6 months of an MI is riskier than if it is performed 6 months after an MI, and surgery within 3 months is associated with a 6% to 30% perioperative infarction rate depending on the quality of monitoring and overall anesthesia care.[27] Likewise, surgery between 3 and 6 months after a previous MI entails a 2% to 15% perioperative infarction risk.[15] Mortality of patients who have a perioperative MI is 40% to 70%.[7, 12]

The urgency of surgery, relative risks and benefits of the procedure, and the patient's status should be considered carefully if surgery is indicated after a recent MI. Although life saving surgery must be performed regardless of risk, elective surgery should be delayed for 6 months.[9] A recent study of patients who had emergency peripheral vascular surgery within 6 weeks of an acute infarction, however, re-

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vealed a 17% incidence of cardiac complications, 12% mortality rate,[30] and 66% ultimate limb salvage. If surgery is semi-emergent, for example, a resectable malignant lesion, a rational approach is to evaluate the patient's functional capacity with studies such as a submaximal exercise tolerance test. If the patient has reasonable exercise capacity, one can consider proceeding with surgery at approximately 6 weeks. Scar formation after an MI is completed between 4 and 6 weeks. Conversely, if the patient is symptomatic during minimal exercise or has major electrocardiogram changes, cardiac catheterization or thallium dipyridamole scan followed by revascularization is recommended.[9]

Angina Pectoris

Exercise tolerance and frequency of angina should be documented in patients with chronic stable angina. Patients should be asked if they can perform common activities such as carrying a 10- to 15-kg object (e.g., grocery bag) up a flight of stairs without having to stop because of chest pain. Ability to do this implies that the functional status is either class I or II. These patients can increase their heart rate and blood pressure to a range that might occur intraoperatively. If patients cannot perform this task, further evaluation is warranted.

Congestive Heart Failure

The degree of left ventricular dysfunction as judged by clinical evidence (New York Heart Association Functional Classification) or by radiologic aids is an important indicator of risk. Low ejection fraction is the most sensitive indicator of ventricular dysfunction, followed by wall motion abnormalities, low compliance, low cardiac output, and high end-diastolic pressure.

Perioperative mortality-morbidity is increased markedly when a displaced point of maximum impulse, presence of 3rd or 4th heart sound, râles, jugular venous distension, or pulmonary edema is demonstrated. A low preoperative ejection faction (EF) correlates well with short-term and long-term survival. One year mortality is significantly higher (30%) in patients with an EF < 0.4.

Hypertension

An association between hypertension and ischemic heart disease has been well-established; hypertension is a major coronary risk factor.[19] Prolonged periods of sustained hypertension accelerate atherosclerosis and lead to variable amounts of end-organ damage in the brain, eyes, heart, kidneys, and peripheral vessels. In addition to decreasing coronary blood flow and oxygen delivery, hypertension increases myocardial oxygen requirements by increasing the afterload and left ventricular mass. Coexisting hypertension increases the occurrence of postoperative MI in patients with a history of MI. In preoperative evaluation of patients with hypertension, adequacy of blood pressure control and drug compliance should be determined. Evidence of end-organ dysfunction must be sought. Only emergency surgery should be undertaken in patients with uncontrolled hypertension (diastolic > 110 mm Hg). Patients who remain hypertensive before induction of anesthesia have an increased incidence of hypotension and evidence of myocardial ischemia on electrocardiogram during anesthesia maintenance.[23] Blood pressure reduction from preinduction values during anesthesia is greater in hypertensive than in normotensive patients.[14] During carotid endarterectomy coexistence of inadequately controlled hypertension has been shown to be associated with an increased frequency of both transient and permanent neurologic deficits.[1] Antihypertensive agents should be continued throughout the perioperative period, as there is no evidence that perioperative antihypertensive therapy adversely influences the conduct of anesthesia.

Dysrhythmias

Unifocal premature ventricular contractions occurring at a rate greater than 10 per 1000 beats, multifocal premature ventricular contractions (PVCs), and R on T phenomenon signify the presence of multivessel disease or significant ventricular dysfunction (EF < 0.4).[20] The 1-year mortality rate in these patients approaches 40% in contrast to 2% in patients with no premature ventricular contractions and an EF > 0.4.[19] Within the first 5 days of acute MI more than 90% of patients develop PVCs. The presence of complete heart block, bundle branch block, and second degree heart block (Morbitz II) also increase risk in the perioperative period.[20]

The incidence of perioperative dysrhythmias is increased in patients with heart disease and a history of preexisting dysrhythmias.[20] Data have been pooled from 40 published studies describing long-term survival of healthy patients who had abnormal or normal electrocardiogram findings. Frequent PVCs, left axis deviation, left ventricular hypertrophy, and ischemic changes are associated with a small but significant risk of death. Nonetheless, evidence does not support the use of a screening electrocardiogram in men without evidence of cardiac disease.

Diabetes

Whether the presence of well-controlled diabetes mellitus increases perioperative risk is debated. Many studies have rejected diabetes as a major cardiac risk factor.[18, 24] The presence of diabetes, however, affects perioperative management. Detection of coronary artery disease is difficult in diabetic patients because MI and ischemia may be painless.[5] Diabetes also increases the risk of clinical atherosclerosis, microangiopathy, and surgical-nonsurgical infections. Survival rates after MI in diabetic patients are lower than in the general population, and the infarct size is bigger.[29]

Valvular Heart Disease

Aortic stenosis is a significant risk factor for poor outcome; the incidence of perioperative cardiac death is increased 14-fold.[12] Clinically it is difficult to differentiate between the murmur of significant

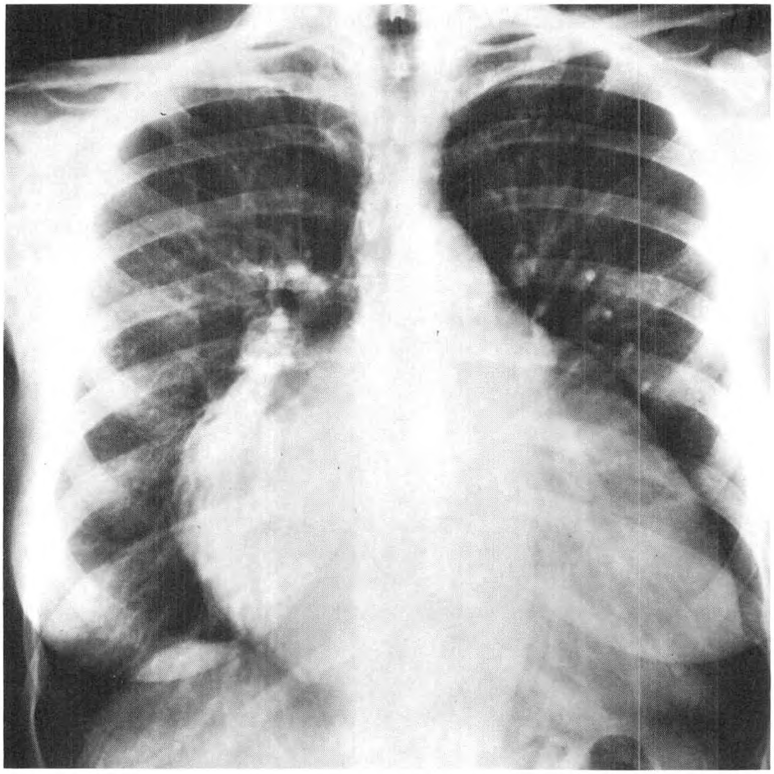

Figure 1. This young woman has pure mitral stenosis that has led to right ventricular enlargement and pulmonary artery hypertension. A chest radiograph such as this one demonstrating cardiomegaly secondary to valvular lesions does not give much information as to adequacy of myocardial function as the enlargement occurs early in the pathologic process.

aortic stenosis and a benign systolic ejection murmur. Precise perioperative evaluation of any suspicious murmur is important. A recent study of noncardiac procedures in patients with moderate to severe aortic stenosis reported that uneventful surgery may be expected in approximately 80% of patients.[33] In the others, transient hypertension or hypotension may occur. In this series, there were no deaths related to the valvular disease. Aortic incompetence and mitral stenosis or regurgitation are associated with little increased risk in the absence of ventricle dysfunction, dysrhythmias, pulmonary hypertension, and coronary artery disease (Fig. 1). All four valvular lesions, however, are associated with an increased risk of postoperative congestive heart failure.[6] In some patients with mitral valve disease, postoperative systemic embolism may occur, and early anticoagulation therapy should be considered.

Mitral Valve Prolapse

Mitral valve prolapse (MVP) occurs in 10% to 15% of the US population. It occurs predominately among young women but without sex discrimination in older individuals. Familial transmission is common (primary MVP). Secondary or acquired MVP is associated with coronary heart disease, cardiomyopathy, left ventricular aneurysm, atrial septal defect, or idiopathic hypertrophic subaortic stenosis.

In MVP the mitral valve is structurally abnormal with elongated chordae tendinae and redundant mitral leaflets. On histologic examination, myxomatous degeneration is seen. An association between Marfan syndrome (4%) and MVP also is recognized.

The pathophysiology of MVP involves the prolapse of the redundant mitral leaflets into the left atrium during midsystole to late systole as the ventricle empties. With reduction in ventricular volume, the degree of prolapse of the mitral leaflets is accentuated. Prolapse also is increased by factors that effectively reduce ventricular volume. Such a condition occurs during increased myocardial contractility, decreased afterload, decreased preload, and tachycardia. Factors that can contribute to MVP include various drugs, Valsalva's maneuver, or a stress response. Conversely, increased left ventricular volume, decreased contractility, increased peripheral resistance, and bradycardia reduce the degree of prolapse. The diagnosis is made when a midsystolic click and late systolic murmur that shifts in timing with posture is detected. Diagnosis can also be made on the basis of late systolic prolapse on M-mode echocardiogram or clear cut billowing of the leaflets in the parasternal long axis view on a two-dimensional echocardiogram.[8] Two groups of MVP have been identified: the classic form of prolapse with leaflet thickening and redundancy and prolapse with thickening but no redundancy. Complications are more in the first group, but the stroke rate is the same in each (approximately 6%).[22]

There is a proven association of MVP with infective endocarditis, hemodynamically important mitral regurgitation, rupture of mitral valve chordae tendinae, neurologic ischemic episodes, and perioperative atrial, junctional, and ventricular arrhythmias.[3, 32] The risk of complications of mitral prolapse (specifically subacute bacterial endocarditis) is greatest in men, older individuals, and patients with mitral regurgitation. Antibiotic prophylaxis is suggested.

When selecting anesthetic agents, the pathophysiology of MVP should be considered even though most patients have minimal hemodynamic consequences. Agents that reduce end-diastolic volume by decreasing both preload and afterload as well as agents that increase myocardial contactility or heart rate exacerbate the degree of prolapse and the incidence of dysrhythmias; they should be avoided. Ketamine hydrochloride, gallamine atropine, sympathomimetic drugs with B effects, and pancuronium bromide are some examples of these agents.

Table 1. *New York Heart Association Angina Classification*

CLASS	DESCRIPTION
1	Angina with strenuous exercise
2	Angina with moderate exercise
3	Angina after climbing one flight of stairs or walking one to two blocks
4	Angina with any activity

Blood volume should be maintained and an extreme head-up position avoided. To reduce the effects of positive intrathoracic pressure on cardiac output, the ventilatory pattern should be one of slow inspiratory flow.

Halothane causes myocardial depression and decreases cardiac output. It also increases the diastolic filling time by both slowing the heart rate and increasing the end-diastolic volume. Thus, halothane might appear to be a good anesthetic choice in patients with MVP, but if there is any evidence of potential decompensation, anesthetic agents with minimal myocardial depressant results should be used. Isoflurane causes little myocardial depression, but the tachycardia it causes may be hazardous for these patients.

Smoking

Continued smoking of tobacco has been shown to triple mortality rates in 11 years in patients with peripheral vascular disease. Patients with chronic obstructive pulmonary disease are also at increased risk of respiratory complications during the postoperative period.

BEDSIDE ASSESSMENT

In most patients an existing cardiac pathologic condition already has been diagnosed. Occasionally the anesthesiologist identifies a cardiac abnormality and must arrange further investigation or referral to a cardiologist.

History

Obtaining an in-depth clinical history is probably the most important component of preoperative assessment. The objective is to grade the level of functional capacity, assess the underlying pathologic condition, and ascertain whether the disease process is improving, static, or worsening. Included is a drug history and record of compliance with prescribed medications as well as an evaluation of hypertension, ischemic heart disease, rhythm disturbances, and diabetes. Four cardinal symptoms of cardiac disease are chest pain, dyspnea on exertion, palpitations, and fainting.

Chest Pain. The New York Heart Association has developed a classification for angina (Table 1). Ischemic pain typically is felt in the lower sternum area, is gripping, aching, or pressing in quality, occurs with exertion or during meals, and is relieved by rest or nitroglycerine. Classicially, it lasts from 1 to 15 minutes; a longer duration suggests unstable angina or MI. Pain may radiate to the left arm, shoulder, jaw, back, or epigastrium. Pain associated with Prinzmetal angina principally occurs at rest and usually is not associated with physical exertion or emotional stress. The electrocardiogram shows ST elevation. The syndrome is associated with a greater incidence of dysrhythmias and conduction abnormalities. Unstable angina is characterized by a progressively worsening angina in intensity or duration and is less responsive to the usual therapy or rest. A new onset chest pain within the last 30 days and transient ST-T wave changes with chest pain also are included in the definition of unstable angina. Unstable angina places a patient at greater risk of developing perioperative complications such as MI.

Location and duration of anginal chest pain has not been correlated with the degree of coronary artery disease or the number of vessels involved. Myocardial ischemia may be totally asymptomatic. It can be detected on exercise stress testing and is associated with a significant decrease in coronary blood flow. The perioperative incidence of silent myocardial ischemia is unknown. Up to 85% of postoperative infarctions are painless and silent myocardial ischemia may indicate that the patient is at risk for an infarction.

Dyspnea on Exertion. Dyspnea is an awareness of breathing that is uncomfortable to the patient, and in the context of cardiac disease it is a symptom of left or right ventricular failure. Ventricular dysfunction correlates well with the amount of exercise that precipitates dyspnea. A careful history, inquiring for symptoms of dyspnea at rest and during exercise, orthopnea, or paroxysmal nocturnal dyspnea enables a more accurate risk assessment to be made. Clinical and radiologic evidence of left ventricular failure in patients with coronary artery disease is associated with poor prognosis. The functional classification described by the New York Heart Association (Table 2) is used routinely to assess left ventricular function. It covers patients with left ventricular dysfunction, dyspnea, and fatigue only at high work loads as well as those whose symptoms at rest prevent any physical activity.

Fainting Attacks. A history of fainting must be investigated preoperatively. Paroxysmal tachycardia, second and third degree heart blocks, aortic stenosis, and cerebrovascular encephalopathy are a few of the causes of fainting in patients with cardiac disease.

Palpitations. The sudden onset of transient palpitations suggests a supraventricular tachydysrhythmia. Longer lasting symptoms of the awareness of heart beats may result from atrial flutter or fibrillation.

Drug Therapy. Recent and past drug ingestions and compliance with prescriptions should be noted. An indication of pathophysiology may thus be obtained.

Physical Examination

There may not be any abnormal physical findings in patients with ischemic heart disease, and the diagnosis may rest entirely on history.

Table 2. *New York Heart Association Functional Classification and its Correlation with Postoperative Mortality*

CLASS	DESCRIPTION OF NYHA FUNCTIONAL CLASS	POSTOPERATIVE MORTALITY (%)
1	Patients with cardiac disease but without resulting limitations of physical activity. Ordinary physical activity does not cause cardiac symptoms.	4.3
2	Patients with cardiac disease resulting in slight limitation of physical activity. Comfortable at rest but ordinary activity produces cardiac symptoms.	10.6
3	Patients with cardiac disease resulting in marked limitation of physical activity. Comfortable at rest but less than ordinary activity produces cardiac symptoms.	25.0
4	Patients with cardiac disease resulting in inability to carry on any physical activity without discomfort. Symptomatic at rest.	67.0

Examination should begin with general observation for cachexia (chronic severe heart failure), obesity, presence of peripheral and central cyanosis, breathlessness, pallor (anemia), clubbing (cyanotic heart disease), splinter hemorrhages (endocarditis), and the presence of visible pulsations over the chest wall. Preliminary assessment of the rate and rhythm of the pulse in both radial arteries should be made and compared to femoral pulses. The carotid pulse rate, rhythm, pulse volume, and character should be noted. Blood pressure should be taken in both arms and in the supine and upright position. Jugular venous distention is a sign of right ventricular failure. In a 45-deg reclining position, the vertical height of the venous column above the level of the sternal angle is the jugular venous pressure. It is normally less than 3 cm H_2O.

Examination of the heart begins with inspection of the chest for asymmetry and the presence of abnormal pulsations. Palpation for the apex beat, heaves, and thrills follow. Lateral and downward displacement of the apex beat suggests cardiomegaly and is associated with a reduced EF.[20] In patients with a previous MI, left ventricular aneurysm, or a hypokinetic segment, there may be an associated abnormal precordial movement, such as a precordial systolic bulging, that is best appreciated by bimanual palpation of the cardiac apex with the right hand while the left hand is placed at the sternal edge. A third heart sound, best heard with the bell of the stethoscope at the apex, arises with rapid filling of the left ventricle when left ventricular end-diastolic pressure is raised. This usually indicates significant ischemic heart disease and MI. A fourth heart sound occurs secondary to atrial contraction, when blood is emptied into a high pressure chamber, with decreased compliance indicating previous MI or ischemia. Auscultation of the neck (for carotid bruits or radiation of an aortic systolic murmur) and the entire precordium for valve murmurs should be done during both phases of respiration. Liver tenderness, presence of ascites, and pedal and sacral edema point toward right ventricular failure.

LABORATORY INVESTIGATIONS

Several tests are indicated.

Complete Blood Count

Ischemic heart disease per se has no effect on hematologic parameters. Elevated hemoglobin levels may be a response to arterial desaturation from right to left shunt or chronic obstructive pulmonary disease in chronic smokers. Polycythemia with hyperviscosity syndrome may lead to ischemia and occlusion. Compensatory tachydysrhythmia with a low hematocrit level may cause angina.

Blood Urea Nitrogen (BUN) and Electrolytes

Diuretic drugs such as furosemide and the thiazides tend to increase potassium excretion. Low potassium levels increase the risk of perioperative dysrhythmias especially if the patient receives digoxin and has significant myocardial dysfunction. Hypokalemia adds to the anesthetic problems of managing patients with low ejection fraction, ventricular dysrhythmias with multifocal ectopic pacemakers, ventricular akinesia, and cardiac failure.[33]

Cardiac Enzymes

Cardiac enzymes are released into circulation after myocardial necrosis. Within the first 12 hours an acute rise in creatinine phosphokinase and associated MB isoenzymes takes place. The level of the MB fraction of the CPK correlates well with the infarct size.[16] Lactate dehydrogenase levels generally increase later to peak at the third day of infarction. Serum glutamic-oxaloacetic transaminase increases within 12 hours, reaches its maximum level at 24 hours, and returns to normal in 3 days.

Liver Function Tests

Although they are usually within normal limits, even in the relatively severe cardiac disease, liver function test results may be abnormal in chronic right ventricular failure. Specifically, enzyme levels are elevated.

The Chest Radiograph

The requirement of routine preoperative chest radiography in patients less than 40 years old who are having noncardiac surgery is controversial.[28] In selective disease states posteroanterior and lateral chest radiographs can provide useful information and indicate cardiac enlargement or increased vascular markings.

An abnormal chest radiograph in patients with coronary artery disease is predictive of myocardial dysfunction, whereas a normal radiograph is not. Abnormal signs that signify myocardial dysfunction are (1) cardiothoracic ratio of more than 50% (normal, < 45%), (2) increased total heart volume or left ventricular volume, and (3) signs of pulmonary edema. MI cannot be detected by chest radiographs unless there is a large aneurysm or calcification.

A normal chest radiograph in a patient with valvular heart disease almost certainly ensures normal ventricular function. The clinical significance of this observation is that specific anesthetic techniques, extensive invasive monitoring, and major therapeutic interventions may not be necessary. An abnormal chest radiograph in a patient with valvular heart disease can occur with either good or poor left ventricular functioning that is caused by early atrial and ventricular enlargements in the presence of good ventricular function.[21] Mitral stenosis is associated with normal left ventricular size, but the left atrium and right ventricle may be enlarged. Mitral regurgitation results in both left atrial and left ventricular enlargement and eventually leads to pulmonary hypertension and right ventricular enlargement. Aortic regurgitation results in elongation and dilatation of the left ventricle. Dilated cardiomyopathy usually is accompanied by significant cardiomegaly and radiologic features of pulmonary hypertension. Hypertrophic cardiomyopathy produces left ventricular enlargement and left atrial enlargement.

Electrocardiography

The resting electrocardiogram may be normal in 25% to 50% of patients with ischemic heart disease. It is rarely normal in patients with significant left ventricular dyssynergy and is the most important preoperative test for patients with ischemic heart disease. Acute ischemic changes include ST depression and T-wave inversion. Early changes of infarction are seen as ST elevation with reciprocal ST segment depression in leads facing away from the site of infarction. Q waves are pathologic when the duration exceeds 0.04 seconds and the amplitude is greater than one-third the height of the R wave from the isoelectric point. MI may occur without Q waves—the so-called non–Q-wave MI. These infarcts may be prognostic of poor outcome.

Ambulatory electrocardiogram monitoring may increase the yield of dysrhythmias and ST-segment changes significantly. There is a good correlation between ST depression during normal activity and abnormal angiographic findings.

The Exercise ECG. Exercise treadmill testing assesses the patient's exercise tolerance, confirms the presence of ischemic heart disease in patients with atypical chest pain, indicates the progression of ischemic heart disease when prior results are compared, and judges the effectiveness of the pharmacologic treatment. It is a relatively inexpensive, safe test with a morbidity rate of 4 per 10,000. The mean arterial blood pressure rises during exercise despite a significant reduction in systemic vascular resistance. Cardiac output increases significantly primarily because of an increase in heart rate, although stoke volume also increases to a small extent (maximum rise, 20%). The systolic ejection period decreases and to maintain normal end-systolic pressure and volume, contractility and wall tension must increase. Because of the increase in heart rate, contractility, and wall tension oxygen consumption increases fourfold with maximum exercise. A reduction in coronary reserve in patients with ischemic heart disease results in myocardial ischemia and dysrhythmias.

Exercise is induced using treadmills to increase myocardial work progressively. Each 3-minute stage in the modified Bruce protocol, which is the most commonly used exercise protocol, corresponds to a number of metabolic equivalents. Both the speed of the treadmill and the gradient are altered. Stage 1 has a speed of 1.7 mph with a 10% grade. Stage 5 has a speed of 5 mph and an 18% grade. Patients who can perform five metabolic equivalents of exercise appear least likely to have a postoperative MI or die. Patients with moderately severe coronary artery disease typically exercise to stage 3 or 4 before stopping because of symptoms or heart rate limitation. This test has significant prognostic value when ST changes are characteristic. The normal ST segment during exercise is steeply sloped, returning to the baseline value within 1.5 small squares (0.06 seconds) (Fig. 2). Downsloping or horizontal ST segment depression of ≥1 mm 0.08 seconds after the J point is diagnostic of ischemic heart disease. Less specific but still indicative of ischemia is an upwardly sloping ST depression of ≥2 mm that lasts more than 0.08 sec after the J point (Fig. 3). Early positive ST changes (within stages 1–3), changes sustained into the recovery period, subnormal increases in heart rate and blood pressure, and ST abnormalities accompanied with angina or dysrhythmia increase the specificity of the test.[11] The electrocardiogram lead in which these changes first appear should be monitored for perioperative ischemia detection. Heart rate and blood pressure values at which ischemic events occur should not be exceeded perioperatively.

Nuclear Angiography

Three different techniques of nuclear angiography can be used.

Thallium Scintigraphy. Thallium scintigraphy can be substituted for preoperative coronary angiography in patients with peripheral vascular disease who have a high risk of ischemic heart disease but are unable to withstand maximum exercise testing.[2] An analogue of potassium, thallium 201, is absorbed by normal myocardium in proportion to local blood flow. Areas of ischemia, previous MI, and acute infarction with an infarct size >5 g appear on the scintigram as filling defects. If these cold spots fill slowly (thallium redistribution) those areas of myocardium are considered ischemic; if they do not fill at all, they are infarcted. A pharmacologic approach has been suggested in which a coronary vasodilator, dipyridamole, is injected to stimulate maximum exercise. After two additional minutes when the dipyridamole effect is considered maximal, thallium 201 is injected. Thallium redistribution seen on the serial images after dipyridamole injection is a marker for relative hypoperfusion of viable myocardium. Patients with positive results of the scan have a 50% chance of de-

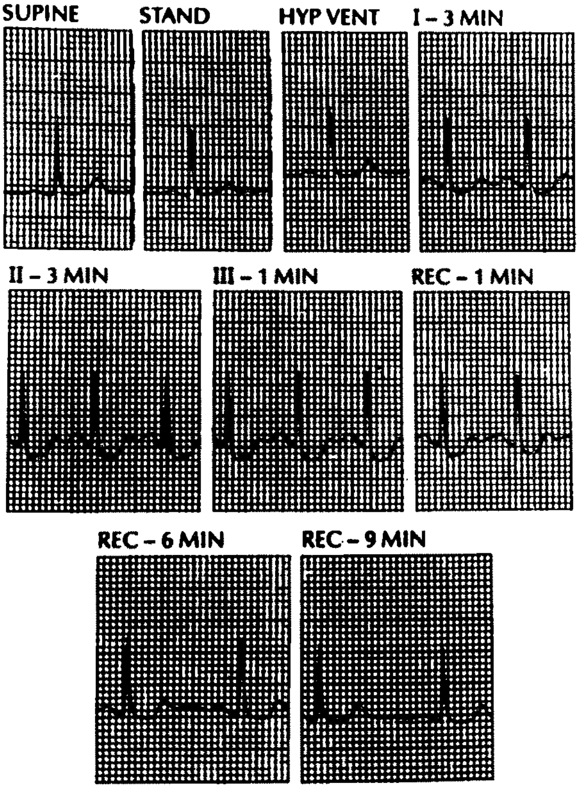

Figure 2. Normal exercise electrocardiogram in a 33-year-old healthy man. Steep slope and quick return to baseline appearance of ST segments is characteristic (scooped STs).

veloping significant cardiac complications after major surgery. This risk can be reduced by percutaneous angioplasty or coronary artery bypass graft surgery before proceeding with peripheral vascular surgery. Combining clinical and thallium data optimizes preoperative assessment of cardiac risk before major vascular surgery.[10]

Technetium Pyrophosphate Imaging. Technetium 99m pyrophosphate is assimilated in infarcted myocardium and is useful to detect MI when symptoms, electrocardiogram results, and cardiac enzyme studies are equivocal or the patient has a left bundle branch block or Wolff-Parkinson-White syndrome. The test result becomes positive 12 hours after MI and remains so for 48 to 72 hours.

Radionuclide Ventriculography. Autologous blood or albumin is labeled with technetium 99m. Gamma radiations emitted by various chambers of the heart are counted either immediately after injections of the isotope (first pass imaging) or during a large number of cardiac cycles (multigated blood pool equilibrium technique). This technique is used to detect intracardiac shunts, regurgitant flows, and right ven-

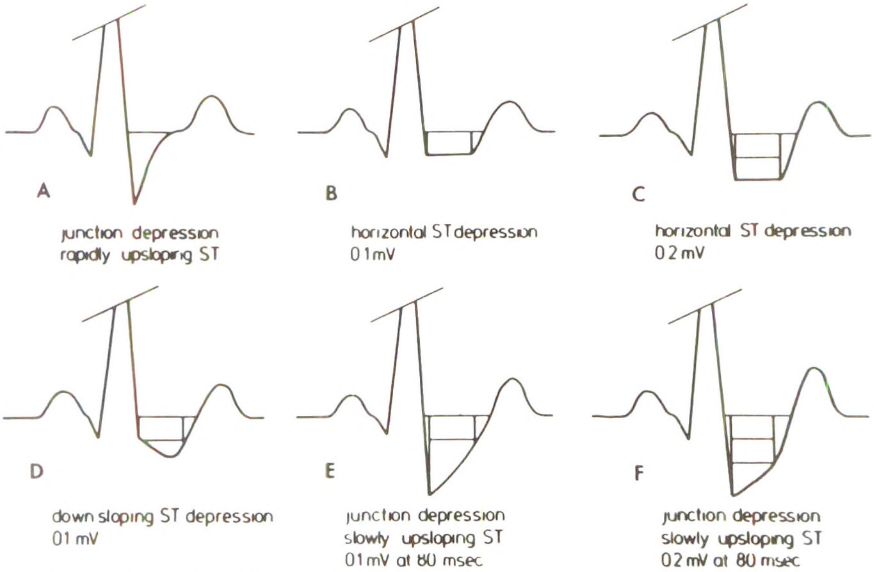

Figure 3. Normal (*A*) and abnormal (*B to F*) patterns of ST segment during exercise electrocardiography are shown. Down sloping, horizontal or up sloping ST segment with depression of more than 1 mm 0.08 seconds after J point suggests the presence of ischemic heart disease.

tricular ejection fraction as well as for exercise testing. A fall in EF after exercise to a rate pressure produce of 25,000 is a more sensitive index of coronary artery disease than exercise electrocardiography.[4]

Echocardiography

The use of multiple beams of sound to construct larger, spatially correct images of the heart enables measurement of end-diastolic and end-systolic chamber dimensions. From these values, ventricle volume, cardiac output, EF, mean circumferential fiber shortening rate, and systolic time intervals can be determined. Correlation between echocardiographic data and angiographic determinations of these variables is good. Two-dimensional echocardiography also can be used to estimate severity of valvular stenotic and regurgitant lesions with great accuracy.

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Preanesthetic Assessment of the Patient with Peripheral Vascular Disease

*Anil M. de Silva, MD**

Major vascular surgery has a relatively high degree of perioperative morbidity and mortality. The process whereby major blood vessels are clamped, sutured, and otherwise manipulated causes significant physiologic derangements and adverse hemodynamic effects. Although peripheral vascular disease itself contributes to patient morbidity and mortality, it is the concomitant disease processes so prevalent in this patient group, that is, coronary artery disease, diabetes, hypertension, renal insufficiency, and poor nutritional status, that have a greater bearing on the perioperative course. A careful preanesthetic assessment of end-organ reserve and a knowledge of outcome studies can help delineate the degree of risk facing each patient.

PATHOPHYSIOLOGY

The term arteriosclerosis encompasses three different vascular disease processes: each contributes to a narrowing and noncompliance of the vascular tree. Atherosclerosis is the predominant form of arteriosclerosis and is characterized by the formation of cholesterol-rich plaques within the intima of the vascular lumen. Degenerative changes occur in the media and adventitia of these vessels. The remaining two forms of arteriosclerosis are calcific sclerosis and arteriolosclerosis. Calcific sclerosis is characterized by deposition of calcium within the media of the muscular arteries, whereas arteriolosclerosis shows hyaline formation within small vessels.[1]

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OUTCOME STUDIES

Atherosclerosis most often affects aortic, cerebral, coronary, and lower limb vasculature. Diabetes, smoking, and hypercholesterolemia appear to accelerate the arteriosclerotic process. Varying degrees of mortality may be ascribed to major vessel disease that is primarily dependent on location of the atheromatous plaques. Type 1 is disease of the aortoiliac vessels: plaque formation is found in the area around the aortic bifurcation and in the common iliac vessels. Type 2 is a more diffuse form of aortoiliac disease and is often found among tobacco smokers who are diabetic and hypertensive. Type 3 is small vessel disease and affects the femoral, popliteal, and tibial arteries.[9] The 5-year survival rates after surgery decline as disease becomes more diffuse: Type 1 disease has a 90% survival rate, type 2 disease an 80% survival rate, and type 3 disease a 65% 5-year survival rate.[21]

Other factors influencing survival are patient's age, extent of coronary artery disease, whether the operation is elective or emergent, and other coexisting disease processes. Elective peripheral vascular surgery (PVS) has an overall mortality rate of approximately 3.0%. An emergent operation, on the other hand, carries a mortality rate of up to 37%.[9, 21]

Studies on aortoiliac reconstruction show that the patient's age has an exponential effect on morbidity and mortality. After the fifth decade of life morality rates increase about 2.3 times per decade. Thus, the mortality rate of a 90-year-old is 28 times greater than that of a 50-year-old.[6]

The presence of coronary artery disease (CAD) is a significant factor in conducting outcome studies. Results of coronary angiography have shown that 60% of all patients who have PVS have significant CAD, and an additional 32% have some disease. Whereas the overall mortality rate for patients having PVS is 3.0%, the rate for patients who had previous cardiac bypass grafting is only 1.5%. Therefore it appears that at least 50% of all perioperative deaths after PVS have a cardiac origin. The mortality rate of those patients with significant CAD who do not have cardiac bypass surgery before PVS is 12%, whereas that of patients with inoperative CAD followed by PVS is 14%.[15]

Of importance in assessing the degree of patient risk versus potential benefit is the effectiveness of PVS. Up to one third of all patients with infrainguinal disease have only partial relief of symptoms after PVS (Table 1).[20]

PREOPERATIVE ASSESSMENT

Cardiac

More than 90% of patients having PVS have CAD, and more than 50% of deaths have a cardiac origin. Clearly, a complete cardiac eval-

Table 1. *Peripheral Vascular Surgery Mortality and Morbidity Rates*

CATEGORY	5 YEAR (%)	IMMEDIATE (%)
Type 1 disease	90	—
Type 2 disease	80	—
Type 3 disease	65	—
Elective	—	3
Emergent	—	37
Previous CABG	—	1.5
Significant CAD, no CABG	—	12
Significant CAD, unable to CABG	—	14

CAD = coronary artery disease; CABG = coronary artery bypass graft.

uation is necessary. An informative cardiac assessment must begin with the patient's cardiac history. Determination of the patient's sleeping patterns, activity levels, medications, smoking habits, family history, and myocardial infarction (MI) or anginal history are all crucial. A physical examination that shows the presence of congestive heart failure or jugular venous distention requires appropriate medical management to improve cardiac function preoperatively. Auscultation that reveals a third heart sound (S3) may indicate an extensive MI; the presence of a fourth heart sound (S4) indicates the possibility of a prior MI. A laterally displaced precordial heave indicates extensive cardiomegaly and an ejection fraction that may be less than 50%.[18] More invasive techniques such as multigated blood-pool imaging and thallium persantine scanning can help determine the extent of cardiac disease better.

In 1977 Goldman et al[12] published their work detailing preoperative factors that might predict cardiac complications. The nine risk factors they believed were most clearly linked to life-threatening cardiac complications were the presence of an S3 or jugular venous distention; MI within the past 6 months; more than five premature ventricular complexes per minute; cardiac dysrhythmias; age greater than 70 years; an intrathoracic, intraperitoneal, or aortic operation; aortic stenosis, emergency operation, and a generally poor medical condition ($Pa_{O_2} < 60$, $P_{CO_2} > 50$, $K < 3.0$, $BUN > 50$, creatinine $>$ 3.0, and elevated transaminases).[12] Nevertheless, the Goldman multifactorial risk index significantly underestimates the risk when a patient has major vascular surgery. For example, patients with a Goldman classification 3 would have a 14% chance of having a cardiac complication if they have a general surgical procedure, while they have a 38% chance of complication with abdominal aortic surgery.[10]

An MI within the past 6 months is prognostic of poor patient outcome. Goldman and Caldera's original study revealed that this category of patient has a 37% chance of life-threatening or fatal compli-

Table 2. *Reinfarction Rates with Previous MI*

AUTHOR	MI < 3 MO (%)	MI < 6 MO (%)	MORTALITY RATE (%)
Topkins[32]	—	54	70
Tarhan[29]	37	16	54
Rao[24] group 1	36	26	57
Rao[24] group 2	5.8	2.3	36

cations. Patients with a third heart sound or jugular venous distention have a 34% occurrence of such complications.

There have been numerous studies of perioperative infarction rates in patients with previous MI. Topkins and Artusio,[32] in 1964, found that patients with an MI within 6 months of surgery had reinfarction rates of 54%: 70% of these MIs were fatal. Tarhan et al,[29] in 1972, reported similar results. This study showed a reinfarction rate of 37% if surgery took place within 3 months of the previous MI. The rate fell to 16% if surgery was performed within 3 to 6 months. A 54% mortality rate was noted in patients with perioperative infarction.[29] Rao et al's studies,[24] published in 1987, divided patients with previous MIs into a population that served as a control group and another that was extensively and aggressively monitored and treated for hemodynamic instability. Group 1 results were typical of previous studies; the reinfarction rates were 36% within 3 months of an MI and 26% if within 6 months. Fifty-seven per cent of Group 1 patients who reinfarcted died. The Group 2 patients, who were aggressively monitored, had a reinfarction rate of 5.8% if surgery was within 3 months of the MI and 2.3% if within 3 to 6 months of the MI. Of those patients in Group 2 who reinfarcted, 36% died (Table 2).[24]

The existence of ongoing ischemia or a relatively recent MI has a direct impact on patient outcome, yet the standard means of determining such pathologic conditions are far from sensitive. Studies have shown that up to 33% of patients may have a painless MI. There is a 30% incidence of asymptomatic ongoing myocardial ischemia, which is especially prevalent in the diabetic population.[33] The patient's history, therefore, may not necessarily be helpful. A resting electrocardiogram will show the presence of CAD in only 50% of patients with the disease. Of those patients with a prior MI, nonspecific ST-T wave changes will be the only electrocardiogram abnormal result in 30% to 50% of cases.[14] Creatinine phosphokinase-myocardial band (CPK-MB) fractions may be accurate in determining the presence of an acute infarct. When a transmural MI has occurred and Q waves are present on the electrocardiogram, the increased CPK-MB fractions are reliable diagnostic indicators. Nonetheless, an ambiguous history with nonspecific ST-T wave changes on the electrocardiogram decreases the incidence of positive CPK-MB fraction to 84%.[19] A chest radiograph may be helpful in determining decreased left ventricular function by demonstrating a decreased cardiothoracic ratio, increased left ventricular volume, or the presence of congestive heart failure.

An array of highly sophisticated echocardiographic and imaging devices exist to delineate the extent of cardiac and coronary disease. Myocardial perfusion imaging using thallium 201 redistribution has been reported to be 100% sensitive to the occurrence of a perioperative cardiac event.[3] The anesthesiologist must maintain a high index of suspicion about the cardiac reserve of the patient with peripheral vascular disease. A patient who has a high probability of developing a perioperative cardiac event will benefit from appropriate consultation with the surgeon and cardiologist. The surgeon must be asked whether the operation is emergent, urgent, or elective. An elective operation will allow the anesthesiologist whatever time is necessary to best prepare the patient medically and collect all helpful data for his intraoperative and postoperative management. An urgent case will allow only 6 to 8 hours, and an emergent case gives less than 1 hour. If the case is not emergent, the anesthesiologist may ask the cardiologist whether the patient is in the best possible medical condition; any specific questions concerning his cardiac status may be addressed also.

The anesthesiologist also will consider carefully how aggressively he wishes to monitor his patient. Although a blood pressure cuff is considered commonplace it is the arterial catheter to which most anesthesiologists turn when blood pressure monitoring on a continuous basis is necessary. A patient who has unstable angina or an acute or recent MI will benefit from this stringent blood pressure control. Surgical procedure in which extreme changes in afterload are expected, such as an operation on the aorta or iliac vessels, also mandate direct blood pressure monitoring.

If the equipment is extremely sensitive a catheter placed in the arterial tree can measure stroke volume, myocardial contractility, and systemic vascular resistance. But because of fundamental inaccuracies in available transducing systems, and because of simultaneous changes in volume status, systemic vascular resistance, and stroke volume, it is not possible to use the arterial catheter to monitor these hemodynamic parameters. Instead, the anesthesiologist relies on central venous pressure monitoring or Swan-Ganz catheterization.

Central venous pressure monitoring is used to monitor right ventricular function when this ventricle is the clinically critical structure or to measure left ventricular function when there is a correlation between left and right ventricular function. If the left ventricle must be monitored and the central venous pressure is inaccurate, then Swan-Ganz catheterization will be necessary. Indications for Swan-Ganz catheterization are a noncompliant left ventricle, left main coronary lesion, valvular disease, ventricular septal defect, MI < 6 months ago, hemodynamic instability, unstable angina, and an ejection fraction $< 50\%$. Close communication between the anesthesiologist and surgeon, aggressive patient monitoring, and vigilant titration

P24631

of the anesthetic to effect should create conditions for the best possible patient outcome.

Hypertension and Its Significance

Among cardiovascular disease processes, hypertension is the most common, affecting more than 20 million Americans. Fifty per cent of patients with peripheral vascular disease have hypertension. The normal upper value for blood pressure in adults is 140/90. More than 90% of all cases of hypertension are classified as essential hypertension. Untreated hypertension eventually leads to renal failure, congestive heart failure, and other systemic disease.[4] In the 1960s, continued antihypertensive therapy was believed to be detrimental to perioperative patient management as these medications interfere with the normal functioning of the autonomic system. Today the consensus is that the hypertensive regimen should be continued undisturbed throughout all perioperative phases to minimize hemodynamic lability. A 1971 study by Prys-Roberts et al[23] found that the untreated hypertensive patient had an 11% incidence of major complications as compared to a 4% incidence in the control group. A 1979 study by Goldman and Caldera[11] found that patient outcome was not worsened if the diastolic blood pressure was stable and remained below 110 mm Hg provided that close intraoperative and postoperative control was maintained. Preoperative systolic blood pressure showed no significant association with intraoperative morbidity. Rather, the report concluded that aggressive and effective intraoperative hemodynamic control played the major role in determining patient outcome.[11]

Diabetes

Diabetes mellitus is common in patients undergoing PVS. Accelerated atheromatous collections predispose patients to vascular insufficiency. The disease is defined as a high fasting-glucose concentration in the blood. Although in the past it was thought that diabetic patients had more complications than their nondiabetic counterparts, this may not be true. When diabetic patients are compared to a control group with the same degree of end-organ damage, no greater morbidity or mortality is seen.[8]

The degree of end-organ reserve is significantly more important in determining perioperative outcome than blood sugar control per se. Because the diabetic patient has a compromised autonomic nervous system, a preoperative evaluation of its effectiveness is necessary. The presence of orthostatic hypotension and decreased pulse rate variability are helpful in indicating effectiveness. Decreased gastric emptying time in the diabetic patient increases the risk of pulmonary aspiration.[30] The preoperative administration of metoclopramide hydrochloride, 10 to 20 mg by mouth 30 minutes before induction of anesthesia, and an H_2 blocker such as cimetidine, 300 mg by mouth the night before and 300 mg intramuscularly 1 hour before surgery, will be helpful.

Despite the anesthesiologist's interest in maintaining hemodynamic stability in the diabetic patient, blood sugar management should not be overlooked. Anesthetics inhibit all phases of immune function.[7] Because hyperglycemia may increase the risk of wound infection,[13] it is important to control blood sugar levels to remain within a normal range. The preoperative value for blood sugar and urinary ketones will help the anesthesiologist determine the efficacy of the medical regimen and whether the patient is at imminent risk for ketoacidosis. A patient who is acidotic, has a blood glucose level > 400 g/dL, and whose dipstick results show the presence of urinary ketones may have this condition. No elective operation should be performed if the patient may have ketoacidosis. There are various protocols for the operative management of blood glucose, but whatever the management method, a rapid means of determining blood sugar, such as the relatively inexpensive portable glucometer, should be available intraoperatively.

Renal Insufficiency

Diabetes and hypertension predispose the patient to varying degrees of renal insufficiency. Chronic renal failure is characterized by a general decline in the number of functioning nephrons until glomerular filtration rates (GFR) are reduced to a dangerous level. More than 50% of nephrons must be lost before the GFR is considered subnormal.[27] Although a plasma creatinine level (normally between 0.7 and 1.5 mg/dL) generally is a reliable indicator for glomerular function, the reduced muscle mass seen in elderly patients with vascular insufficiency may reduce the sensitivity with which it reflects GFR. Creatinine clearance is a more reliable means of determining glomerular filtration; mild renal disease is present with clearance between 50 and 80 mL/min, and moderate renal disease is present when clearances range from 25 to 50 mL/min. Normal creatinine clearance ranges from 110 to 150 mL/min.

Chronic renal insufficiency indicates significantly less renal reserve than is available normally. Many patients for vascular procedures arrive at the operating room in a dehydrated state because of combination of factors such as a fasting state, poor nutrition, and the preoperative administration of contrast agents for radiographic studies. Misjudging the extent of hydration can precipitate oliguria and lead to acute renal failure, which is associated with mortality rates in excess of 50%.[16] Dry mucous membranes, poor skin turgor, and orthostatic hypotension will alert the anesthesiologist to the patient's intravascularly depleted condition. Fluid resuscitation with balanced salt solutions to maintain urinary output above 0.5 mL/kg/min will help avoid irreversible renal damage. Great care must be taken to prevent overhydration, especially if the patient has a history of congestive heart failure. Invasive monitoring with a central venous or pulmonary artery catheter will help establish an appropriate hydration state. Avoidance of drugs that are nephrotoxic, such as gentamicin, or that are primarily excreted renally, such as pancuronium bromide, probably is wise.

Anemia

Many patients with peripheral vascular disease are chronically anemic. Their malnourished state and their chronic renal insufficiency with the attendant decreased erythropoietic production contribute to their condition.

Anemia ordinarily is not a problem if the patient is in generally good physical health.[2] Although the oxygen content of the blood is depressed there is an increase in 2,3-diphosphoglycerate (DPG) levels, which shift the oxygen-hemoglobin dissociation curve to the right and allow more efficient unloading of oxygen to peripheral tissues. In addition, when hemoglobin levels fall below 8 g/dL there is an increase in cardiac output, which adds to the compensatory response to anemia. This increase in cardiac output, which is secondary to an increase in heart rate, stroke volume, and a fall in systemic vascular resistance, increases myocardial oxygen consumption.[4, 25] The patient with CAD is at risk when these hemodynamic changes occur.

Other studies report that hemodilution to a hemoglobin level of 10 g/dL optimizes oxygen transport.[28] Higher levels of hemoglobin increases blood viscosity, which in turn decreases cardiac output and lowers the overall oxygen carrying capacity. Patient outcome, in general, is better at hematocrit levels of approximately 30%.

Although hemoglobin levels must be considered in the preanesthetic assessment, the transfusion risk also must be recognized. Hepatitis occurs in approximately 7% to 10% of transfused patients. Ninety percent of these patients have non-A non-B hepatitis, and chronic active hepatitis[17] develops in about 50% of those affected. In addition, there is the risk of contracting other blood-borne diseases such as cytomegalovirus and acquired immune deficiency syndrome (AIDS). The use of pedigree blood, autologous blood products, and intraoperative cell saving techniques help limit exposure to pathogens. Transfusion reactions also may occur. The anesthesiologist, therefore, must consider all of these factors before the operation.

Malnutrition

Patients undergoing PVS often are malnourished, perhaps because of a relative inability to ambulate and the consequent decreased oral intake. Several studies show that a malnourished state in the surgical patient increases morbidity and mortality.

Casey et al,[5] in 1983, correlated nutritional status and incidence of wound infections in vascular surgery patients. The incidence of wound infection in patients with serum albumin levels below 3 g/dL was three times greater than that in a control group. Smale et al,[26] in 1981, showed that in malnourished patients, the use of preoperative total parenteral nutrition (TPN) was associated with a threefold decline in mortality.

During the preoperative visit, the anesthesiologist may evaluate the patient's nutritional status by asking about the patient's usual oral intake and whether there is a history of recent weight loss. A visual assessment will help determine whether the patient's body mass index (BMI) is appropriate: BMI = weight in kg/height in m^2. Normal values are between 20 and 30 kg/m^2. A patient with a BMI greater than 20% of the normal value is considered obese; a BMI greater than 50% is indicative of morbid obesity. Albumin levels less than 3 g/dL or a total serum protein level of less than 6 g/dL also indicates nutritional depletion.[4]

If the anesthesiologist suspects malnourishment a consultation with the nutrition specialist is necessary. A 6- to 10-day preoperative course of TPN may be indicated if nutritional reserves are extremely depleted.[26]

SUMMARY

The preanesthetic assessment of the patient with peripheral vascular disease entails a careful examination of the organ systems most affected. Judicious determination of the degree of end-organ reserve can help elucidate the degree of risk, the extent of invasive monitoring required, and indeed, whether the planned procedure is actually warranted. Optimal patient outcome requires close communication between anesthesiologist, surgeon, and any consultants who may be necessary for a complete understanding of the patient's overall medical state.

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Preanesthetic Assessment of the Patient with Respiratory Disease

*Elizabeth A. M. Frost, MD**

Respiratory disease is a common problem encountered by the anesthesiologist during preanesthetic assessment. Cleaner air and a decrease in tobacco smoking by the general population has reduced the severity of chronic obstructive lung disease. Increasing age of the patient population, however, has had the opposite effect.

Adequate preoperative assessment is important to delineate the disease, identify patients at risk for postoperative complications, and outline a preanesthetic therapy plan that may improve lung mechanics. The type of procedure has important bearing on assessment, and discussion with the surgeons is imperative. Rarely must an operative procedure be cancelled because of respiratory disease in chronic or acute situations.

CHRONIC RESPIRATORY DISEASE

The most common chronic respiratory diseases encountered by the anesthesiologist are chronic obstructive pulmonary disease (COPD), lung tumors, and asthma.

In the assessment of these disorders, there is much common ground. Nonetheless, each pathologic process must be evaluated specifically.

Risk Factors

Patients with chronic lung disease and limited pulmonary reserve are vulnerable to reductions in static lung volumes and widening of A-a gradients. This is common in abdominal and thoracic surgery.

Risk factors other than surgery that increase the incidence of postoperative respiratory complications, especially atelectasis, include

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smoking 10 or more cigarettes daily,[45] even when pulmonary function tests are normal,[10] obesity,[37, 58, 61] and anesthetic time over 3 to 4 hours,[13, 37] although this last issue has been disputed.[48] The sex of the patient[64] or the type of anesthetic used[11, 13] do not increase risk when smoking histories are considered. Age per se in the absence of pulmonary changes may or may not increase the risk of postoperative pulmonary complication.[26, 48, 60, 64]

GENERAL HISTORY AND PHYSICAL EXAMINATION

Several essential questions must be answered: (1) Does the patient have a cough? (2) Does the patient expectorate phlegm? (3) Does the patient become out of breath when walking uphill? (4) Does the patient smoke?

If the answers are positive, more questions are posed to determine the timing and amount of coughing, the character of the sputum, the time of dyspnea onset, and the smoking history. Patients with emphysema and bronchitis tend to minimize their symptoms, a fact that must be considered when assessing the degree of pathologic conditions present.

Dyspnea, a subjective sensation caused by a ventilatory effort out of proportion to the physical activity, may result from either pulmonary or cardiac disease. If pulmonary in origin, severe dyspnea implies a significantly diminished respiratory reserve, that is, a forced expiratory volume in 1 second (FEV_1) of 1500 mL or less. A smoking history, quantified in terms of pack-years, is a risk factor for COPD and malignancy. This risk increases with the number of pack-years. A productive cough that lasts at least 3 months each year for at least 2 years indicates chronic bronchitis, which predisposes the patient to cor pulmonale. A history of asthma or wheezing raises the possibility that a reversible airway obstruction may be present. All medications taken must be recorded, as several of these may interact with anesthetic drugs.

The physical examination assesses the degree of airway obstruction and sputum retention. An area of coarse râles on auscultation may indicate bronchiectatic patches; absence of breath sounds may be due to collapse or pleural effusion. Airway obstruction resulting from bronchitis, emphysema, or asthma causes hyperinflation, which is best observed as an increase in the anteroposterior diameter of the chest. The chest appears to remain in an inspiratory position. Hyperresonance may be noted on percussion, and cardiac dullness is obliterated as the hyperinflated lung interposes between the heart and the chest wall to give a resonant note. Wheezing is produced by the high velocity of air jetting through narrowed and collapsed airways. A vital capacity breath may be required before the wheezing is appreciated. Rarely, in severe asthmatics, is wheezing absent if the air flow is slowed to critical levels by the narrowed bronchi. Airway obstruction is indicated further by appreciation of the prolongation of the expiratory phase of respiration. Normal expiration after a forced vital capacity maneuver as auscultated over the larynx lasts 1 to 2 seconds and is clear. As airway resistance increases, this time may be prolonged such that the patient must take another breath before air flow steps. Wheezing is heard easily.

Retained sputum is heard as moist sounds, mostly at the lung bases posteriorly. Disappearance after coughing indicates little degree of bronchial pathologic conditions. Persistence of sputum points to bronchiectasis. If sputum has obstructed bronchi, there is no air entry and no sound.

Laboratory Studies

Examination of a chest radiograph may reveal unsuspected diseases such as carcinoma or tuberculosis. Opacities may indicate bronchopneumonia that requires preoperative treatment. In bronchitis, lung markings usually are prominent, especially in the upper zones (a reversal of the usual pattern). Lung markings are diminished in emphysema, especially at the bases. Bullae may be seen.

CT chest scans are especially useful in evaluating mediastinal lesions, pleural effusions, and the extent of bronchiectasis and emphysema.

A complete blood count may reveal an increased level of polymorphonuclear leukocytes, thus indicating infection. Hemoglobin levels greater than 16 g/dL suggest chronic hypoxia. Results of arterial blood gas analyses may show both physiologic and pathologic responses to chronic lung disease.

Changes in compliance, tidal volume, and frequency are associated with a decreased arterial carbon dioxide tension (Pa_{CO_2}) and increased arterial pH (respiratory alkalosis). Although alveolar ventilation is increased, the arterial oxygen tension is decreased because of continued perfusion of alveoli with small gas volume, the low ventilation-perfusion ($\dot{V}/\dot{Q}$) units. If preoperative therapy accomplishes deep inspiration, lung volume and mechanics improve and physiologic shunt decreases. In patients with long-standing disease and structural changes, Pa_{CO_2} may be elevated and Pa_{O_2} significantly decreased. Hypoventilation may result from chronically raised Pa_{CO_2} or pulmonary mechanical dysfunction (e.g., chronic bronchitis).

The response to increased work of breathing varies: some patients do not increase breathing and thus become cyanotic and hypercarbic (blue bloaters); others increase the work of breathing and have normal Pa_{O_2} and Pa_{CO_2} levels (pink puffers). This latter group is dyspneic and may have more emphysema. Gas transfer defects are found in patients with interstitial lung disease such as sarcoidosis and pneumoconiosis. There are few signs of obstruction, but the patient is dyspneic on exertion. Pa_{O_2} is normal and Pa_{CO_2} is reduced because hyperventilation in response to a lowered Pa_{O_2} excretes more CO_2 from the normal lung, which then compensates for the inadequate CO_2 transfer in the diseased lung. Similar compensation for oxygen is not possible because of the differences in its dissociation curve.[40]

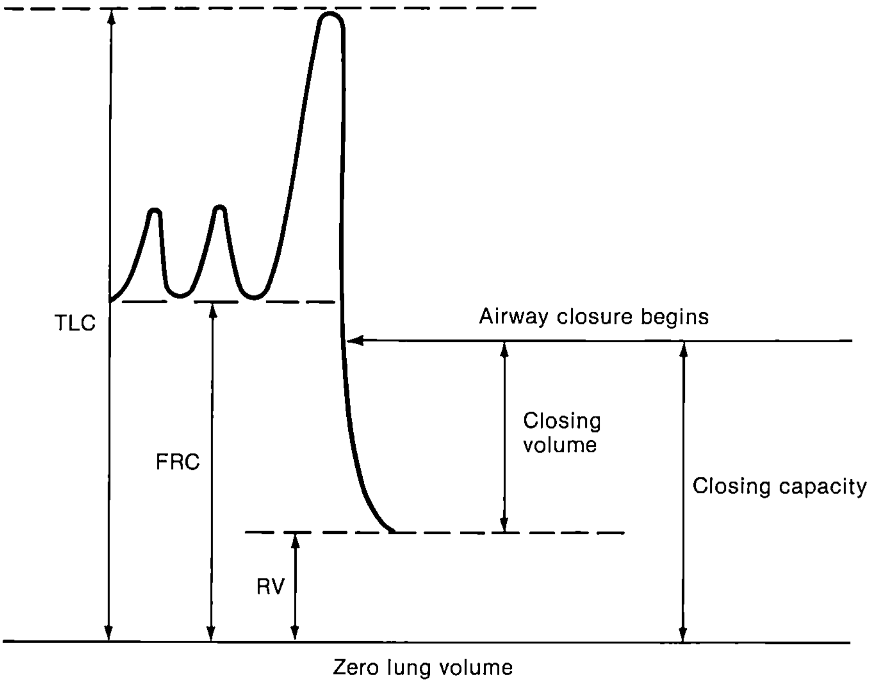

Figure 1. The spirogram illustrates the relationship between closing volume and closing capacity. The example is a young, healthy adult with a closing capacity less than functional residual capacity (FRC). TLC = total lung capacity, RV = residual volume.

Pulmonary Function Tests

Despite the wide use of pulmonary function tests, only a few studies are predictive. Current evidence suggests that spirometry is the most reliable and cost-effective means of evaluating risk. Spirometry is based on a vital capacity or forced vital capacity (FVC) maneuver. The subjects inhale as deeply as possible, then exhale as fully and forcefully as they can. Volume is recorded as a function of time. The maneuver is thus designed to assess flow rates. An additional benefit to the analysis of a flow-volume loop is that it involves both a forced exhalation and a forced inhalation; the latter maneuver identifies extrathoracic upper airway obstruction, which is of particular interest to the anesthesiologist. From the plot of volume versus time obtained during a spirometric maneuver, specific data points are obtained. First, the total volume of lung capacity exhaled into the spirometer is measured, and this is designated as the FVC.

The functional vital capacity is divided into several time intervals (Fig. 1). The most reproducible of these is the FEV_1. Persons with normal lungs should be able to exhale 75% to 80% of FVC within this time, whereas those with increased airway resistance will exhibit re-

Table 1. *Normal Pulmonary Function Test Values for a 50-Year-Old Man, 70 kg, 180 cm, Sitting Position*

TEST	VALUE
FVC	4.74 L
FEV_1	3.87 L
FEV_1/FVC	83%
PEFR	8.97 L/s
MMF	3.08 L/s
D_{co}	32.1
ERV	0.96 L
RV	1.77 L
TLC	6.51 L
RV/TLC	27%
FRC	2.73 L
IC	3.78 L

FVC = forced vital capacity, FEV_1 = forced expiratory volume in 1 second, PEFR = peak expiratory flow rate, MMF = midmaximal flow rate ($FEV_{25\%-75\%}$), D_{co} = diffusion capacity for cardiac output, ERV = expiratory reserve volume, RV = residual volume, TLC = total lung capacity, FRC = functional residual capacity, and IC = inspiratory capacity.

duction in airflow rates and a decrease in both FEV_1 and the FEV_1:FVC ratio. Normal pulmonary function test values are listed in Table 1.

The early portion of the FVC curve is, however, more effort-dependent, and the terminal portion can be recorded poorly (e.g., when the sensation of dyspnea causes the subject to terminate the FVC maneuver prematurely). The midportion of the curve, which is measured between the first and last quarters of the FVC spirogram, is considered the most effort-independent portion and is the most sensitive indicator of small airways obstruction (as in the asthmatic patient). This parameter is known as the forced expiratory flow $(FEF)_{25\%-75\%}$ or as the midmaximal expiratory flow rate (MMEFR). Although other parameters can be generated, these are the most useful for detection of obstructive airway disease.

Characteristics of obstructive pulmonary disease are normal or decreased vital capacity, decreased FEV_1, and decreased $FEF_{25\%-75\%}$. They are evaluated against a set of "normal" numbers based on the patient's age, sex, and height. FEV_1 or $FEF_{25\%-75\%}$ of at least 75% of the predicted value is normal; mild obstructive disease is present at flow rates 65% to 75% of predicted, moderate at 50% to 65%, and severe obstructive disease if flow rates are less than 50% of predicted values.[19]

Once airflow limitation is established, its reversibility is determined. This is done by repeating the spirometry approximately 20 minutes after administration of an inhaled bronchodilator. A significant bronchodilator response requires an improvement of at least 15%

in two of the three parameters, FVC, FEV_1, or $FEF_{25\%-75\%}$. Many factors can affect the response to inhaled bronchodilators, such as the chronicity and severity of airflow limitation, dosage and technique of bronchodilator administration, complete withdrawal of pre-existing therapy, smoking history, age of patient, presence of respiratory infection, and time of day. Therefore, more accurate results are obtained if testing is done in a pulmonary function laboratory where more formalized and extensive testing of patients with respiratory disorders can be done.

Pulmonary tests used that are used rarely include static lung volume measurements either by body plethysmography or gas dilution and serial lung volume measurements that may indicate a bronchodilator response not evident by spirometry. Although flow rates cannot be assessed directly by this latter technique, relief of airway obstruction is inferred from decreases in residual volume and functional residual capacity (FRC).

Flow-volume curves also can be generated during the FVC maneuver. Flow is recorded as a function of volume and provides the same information as the standard spirogram but represents it differently. It is useful, however, in differentiating between diffuse airways disease and a central obstruction. Distinction also can be made between fixed and variable obstruction, and the location, either intrathoracic or extrathoracic, can be pinpointed.

Additional tests include diffusing capacity, direct measurement of airways resistance, measurement of static and dynamic compliance of the lung, and measurements of closing volume that may be useful in screening for subclinical pulmonary disease and may have significant predictive value for the later development of overt disease.

Because clinical studies generally have been given widely different predictive values for the use of pulmonary function tests[34, 41, 51, 55] with the possible exception of use before lung resection, no single values represent an absolue contraindication to general anesthesia.

PREVENTION OF POSTOPERATIVE COMPLICATIONS

Prevention of postoperative atelectasis or the subsequent pneumonia include preoperative identification of patients at higher risk. Although preoperative pulmonary function tests may be useful as outlined,[56] most clinicians believe that a good history and physical examination adequately identify the high-risk pulmonary patient.[40, 55, 56] A history that includes tobacco smoking, presence of recurrent pulmonary infections, occupational background, (e.g., coal miners, construction workers, or those who work with asbestos or on assignment to high-polution areas), asthma, exertion necessary to bring on dyspnea, and presence of chronic bronchitis is significant. If in doubt, a baseline chest roentgenogram and arterial blood gas analysis should be obtained with or without preoperative pulmonary function studies.

Many different methods have been used to prevent postoperative atelectasis.[8, 9, 50] The techniques most widely used are geared toward restoring the restrictive pulmonary deficit with its accompanying increased work of breathing and hypoxemia. The cause of the restriction is related to small tidal volumes with no sigh breaths that over several hours results in a difficult to reverse pattern of atelectasis. To minimize this phenomenon the sigh can be introduced artificially by encouraging the patient to inspire deeply. The deep breath increases transpulmonary pressure and distends the lungs. Studies in dogs and humans have indicated that alveoli, once inflated, remain inflated for at least 1 hour.[17, 32] Therefore, any respiratory maneuver should be carried out hourly to maintain sustained alveolar inflation and maintenance of normal lung volumes. An additional maneuver, sitting in a chair, has been shown to increase FRC compared to the supine position.[33]

The most popular method to improve postoperative pulmonary function is incentive spirometry (IS), and it is used in 95% of hospitals. The next most popular methods are chest physical therapy, which is used in 83% of hospitals, intermittent positive pressure breathing (IPPB) in 82%, continuous positive airway pressure (CPAP) in 25%, and blow bottles in 17%.[42] Randomized studies indicate that deep breathing exercises with[3, 12, 23] or without an adjunctive breathing device (such as incentive spirometry) are superior to no treatment,[60] conventional chest physical therapy,[3, 12] and IPPB.[62] One study looking only at conventional chest physiotherapy with deep breathing versus IS in 103 laparotomy patients revealed a 2% incidence of pulmonary complications with deep breathing exercises alone and a 5% incidence with IS.[2] The general conclusion from these studies is that all deep breathing exercises decrease pulmonary complications.[2] The most popular mode of deep breathing is with a quantifiable IS device, although no specific IS device appears superior.[28] All patients, particularly high-risk chronic obstructive lung disease patients undergoing upper abdominal or chest surgery, should have preoperative instruction in deep breathing exercises before surgery. The ideal deep breath should be one that is held for 3 seconds. A 3-second deep breath is superior to multiple deep breaths or a single, unsustained deep breath.[63] The deep breathing maneuver used should be supervised hourly because preoperative patient education and training alone is not effective in decreasing postoperative pulmonary complications.[47]

Continuous positive pressure mask systems that cover both the nose and mouth can decrease the incidence of postoperative pulmonary complications more than IS alone.[46, 57] Disadvantages are that a more expensive and complex system is required, there is discomfort in tolerating a tight-fitting mask, and the potential for aspiration of gastric contents exists.[54] A more feasible system in the future may be nasal CPAP. Preoperative cessation of smoking may or may not decrease postoperative respiratory complications. Although carboxyhemoglobin levels can be reduced acutely, thus increasing the margin of safety, an increase in viscosity of sputum that occurs shortly after smoking cessation in the absence of improved small airway and ciliary function may actually increase the risk of postoperative problems.

The following routine measures can be taken to minimize postoperative atelectasis.

1. Preoperative screening of high-risk patients by history, physical exam, and possibly chest radiograph, arterial blood gas, and so forth.
2. Preoperative training of high-risk patients in the use of deep breathing exercises of some type.
3. Breathing exercises that emphasize a large inspiratory effort of at least a 3-second duration.
4. Supervision of breathing exercises at least once each hour while awake.
5. Adequate pain relief to allow deep breathing.
6. Encouragement, when possible, to remain in an upright position in bed.
7. Sitting upright in a chair as soon as possible.
8. A trial of mask CPAP or nasal CPAP for refractory atelectasis.

The Asthmatic Patient

Asthma is a common disorder, with estimates of its prevalence in the US population ranging from 3% or 4%[5] to as high as 9% or 10%[16]; the higher figures include self-diagnosis and less stringent criteria. A useful definition is provided by the American Thoracic Society and the American College of Chest Physicians. Asthma is a disease characterized by increased responsiveness of the airways to various stimuli that is manifested by widespread airway narrowing, which changes in severity either spontaneously or as a result of therapy.[49] Various types of asthma are subsumed including extrinsic and intrinsic asthma.

In extrinsic asthma, the disease is provoked by an inhaled allergen (e.g., mold, dust). This type of asthma is IgE-mediated and is associated with atopy, with elevated serum IgE levels, and often with eosinophilia of the blood or sputum. The skin test with an extract of the specific allergen is positive. Extrinsic asthma usually begins in childhood and may disappear with age. Attacks, however severe, tend to be self-limited. Treatment of the acute episode generally suffices, with no need for chronic therapy.

Intrinsic asthma is much more common (up to 50% of all asthmatic patients have intrinsic asthma). No specific immunoprovocative agent can be identified, and it tends to occur in an older age group. The attacks are more severe, more difficult to break, and more often fatal. People with this type of asthma require therapy not only to treat the acute episode but chronically to prevent exacerbation. Manifestations of both intrinsic and extrinsic disease may be found in the same individual.

Additional types of asthma exist, such as those related specifically to exercise, to various agents, and to the ingestion of aspirin and other nonsteroidal anti-inflammatory drugs. Regardless of the cause, the pathologic and pathophysiologic changes are constant, namely dyspnea, chest tightness, and wheezing. There always is a prolonged expiratory phase, and the attack may remit spontaneously, improve with therapy, or worsen over time.

During an asthma attack, the bronchial smooth muscle contracts. Concentric hypertrophy of muscle within bronchial walls may reduce airway diameter further. Bronchoconstriction may be caused by irritant receptors or release of inflammatory mediators, among which histamine, bradykinin, leukotrienes C_4, D_4, and E_4, prostaglandins D_2, F_2 and G_2, adenosine, thromboxane A_2, and platelet activating factors have been identified. As vascular permeability provoked by these mediators increases, plasma proteins leak from capillaries into the interstitium of the lung, and edema results throughout the airways. Leukocytes are attracted, and the bronchial mucosa becomes infiltrated with eosinophils, neutrophils, plasma cells, lymphocytes, and macrophages. Increased production and viscosity of mucus further contributes to luminal narrowing. The clearance of mucus and cellular debris is compromised by desquamation of epithelial cells, with loss of cilia, diminished function of remaining cilia, and replacement of ciliated epithelium with goblet cells. Early in an asthmatic attack, while simple bronchoconstriction is still the major factor in airway narrowing, the attack may be aborted by bronchodilators. As the episode progresses and more of the airflow obstruction is due to mucus plugging, inflammation, and airway edema, such pharmacotherapy is less useful.

Airway narrowing and resistance to airflow affects pulmonary function.[21, 27] The obstruction is nonuniform, so ventilation-perfusion mismatch ensues with an increase in wasted ventilation and a decrease in Pa_{O_2}. Closing volume is increased because high negative intrapleural pressures must be generated to force expiration past a narrowed lumen, hence airways close prematurely during expiration. Air trapping with subsequent hyperinflation of the lungs results. All the static lung volumes are increased, especially functional residual lung capacity and total lung capacity. Indeed, this may be so marked in cases of severe bronchospasm that FRC approximates total lung capacity (TLC), leaving minimal inspiratory reserve.

The increase in FRC, which is also thought to contribute to the increased ventilatory drive seen in acute asthma, maintains airway patency but at great cost. A much higher transpulmonary pressure gradient must be generated with pronounced increase in negative intrapleural pressure. Pulmonary hypertension can result as the right ventricle is pressure- and volume-loaded. The left ventricle is relatively underfilled, and there is an increase in left ventricular afterload.

Treatment. Many drugs are available to treat asthmatic attacks (Table 2). Several reviews have considered the appropriate choice and action of these agents.[15, 20, 24, 52, 53]

Preanesthetic Preparation. Simple bedside spirometry is generally sufficient in the asthmatic patient. Various hand-held devices are available. Many asthmatics patients will be familiar with these devices already; some will even know their now best peak flow.

Consideration must be given to (1) baseline pulmonary function,

Table 2. *Drugs Useful in the Prevention and Treatment of Asthma Attacks*

TYPE	DRUG	ROUTE AND DOSE (ADULT)
Adrenergic agents Activate adenyl cyclase, cAMP		
Mixed and B	Ephedrine	Oral: 25 mg q4–6 hr
	Epinephrine	Subcutaneous: 0.3 mg Inhalation: 0.25–0.6 mg q 2–3 hr
Mixed B_1 and B_2	Isoproterenol	Inhalation: 0.25–0.6 mg q 1–2 hr
B_2 selective	Isoertharine	Inhalation: 0.25–1.0 mg q 4–6 hr
	Metaproterenol	Oral: 1.5–2 mg q 24 hr Inhalation: 1.3–2 mg q 2–4 hr
	Albuterol	Oral: 2.5 mg q 26 hr Inhalation: 0.1–0.2 mg q 4–6 hr
	Terbutoline	Subcutaneous: 0.25 mg q 4–6 hr Oral: 2.5–5 mg q 26 hr Inhalation: 0.2–0.4 mg q 4–6 hr
Methylxanthines[*]	Aminophylline	Oral: 13–16 mg/kg/day titrated to clinical effect and serum level Intravenous: loading dose 5.6 mg/kg, maintenance 0.2–0.9 mg/kg/hr
Anticholinergic agents[†]	Atropine	Oral: 0.4–1 mg q 26 hr Inhalation 25–75 μg/kg q 3–6 hr
	Glycopyrrolate	Intravenous, subcutaneous, or inhalation: 4–5 μg/kg q 2–8 hr
Cromolyn sodium[‡]		Inhalation: 20 mg q 26 hr
Corticosteroids[§]		Oral: drugs and doses extremely variable Inhalation: beclomethasone 80–160 μg q 6 hr

[*] Mechanism unclear: Inhibits phosphodiesterase and increases cAMP, inhibits intracellular calcium activity, inhibits prostaglandin actions, directs antagonism of the adenosine receptor.

[†] Inhibits generation of cyclic GMP, causing bronchodilation; inhibits tracheobronchial secretions.

[‡] Stabilizes mast cells; useful only in prophylaxis.

[§] Mechanisms ill-defined; potentiate response to aminophylline and sympathomimetics; reduce inflammation and airway edema.

Adapted from Plante LA: Preanesthetic assessment of the asthmatic patient. Anesthesiology News 16:2, 1990, p 13; with permission.

(2) current pulmonary function, (3) therapeutic regimen, (4) proposed surgical procedure, and (5) anesthetic options.

Information should be obtained about the duration and severity of the disease. Childhood asthma that has since disappeared is of little importance in the adult. The factors precipitating the attacks, however, should be documented. The frequency and required therapy of attacks also should be noted.

The physical findings in asthma may be both variable and misleading. Surgery should be postponed for a patient who is actively wheezing, if possible, to improve pulmonary function. The absence of wheezing may indicate either the quiescent period between attacks or may indicate marked impediment to airflow. Other physical signs that are indicative of severe bronchospasm include tachypnea, use of accessory muscles of inspiration, pulsus paradoxus, cyanosis, or disturbances of consciousness.

The chest radiograph is more useful in excluding other causes of pulmonary disease than in evaluating asthma. Emphysematous changes, bronchiectasis, tracheal obstruction, or pneumonia may be diagnosed. Both the chest radiograph and arterial blood gas analyses are unlikely to be helpful in the asymptomatic asthmatic.

The electrocardiogram should be reviewed. Evidence of right axis deviation or right ventricular strain (p pulmonale, right ventricular hypertrophy) suggest persistently elevated pulmonary vascular resistance, hence more severe pulmonary disease. The anesthesiologist should also look for tachydysrhythmias or ischemic changes, which may suggest either an acute attack or bronchodilator toxicity.

If patient receives theophylline, serum drug levels should be measured. The therapeutic range for theophylline is generally cited as 10 to 20 μg/mL but much variation is described.[52]

Premedication. Patients taking theophylline orally or β-agonists should continue their regular regimen on the day of surgery. Exceptions to this rule are for those in whom the serum theophylline level is high enough to raise concern about drug toxicity. Those receiving inhaled drugs (β-agonists, cromolyn, ipratroprium, or steroids) should also continue their regular regimen as should those who use metered-dose inhalers only intermittently. Two or three puffs should be given several hours before the anticipated time of surgery. The use of β_2 selective agents is preferred to the nonselective agents such as isoproterenol. The exact timing depends on the individual agent. Metaproterenol, for example, has a duration of action estimated at 2 to 4 hours and should be given closer to the time for surgery than terbutaline, fenoterol, or albuterol, of which the duration for each is 4 to 6 hours. Although any of these drugs can be given as needed in the operating room, other effects (nervousness, tachycardia, or palpitations) may exacerbate anxiety. Patients taking intravenous theophylline should have their dosage decreased by about one third on the morning of surgery and continued intraoperatively at the lower infusion rate. The rationale for this recommendation concerns decreased clearance of aminophylline secondary to diminished liver blood flow while under general anesthesia and the risk of toxic levels of aminophylline developing if the usual doses are continued.[31] Patients taking steroids should receive stress doses perioperatively. Those on inhaled steroids are at much lower risk of adrenal suppression and probably do not require supplementation.

It is commonly believed that anxiety can precipitate bronchospasm in susceptible individuals, although controlled studies are lacking. The preoperative visit by an anesthesiologist to explain the medication and reassure the patient is often sufficient to allay anxiety. For individuals in whom additional preanesthetic sedation is required and who have no signs of impending respiratory failure, almost any of the common premedicants can be used.[18, 25] Diphenhydramine provides both sedation and blockade of histamine-induced bronchoconstriction. Both hydroxyzine and droperidol decrease airway resistance. The barbiturates have been shown to increase bronchial tone in excised lung tissue, but there is no convincing evidence of this in vivo. In asthmatic patients who are sensitive to sulfur, avoidance of thiobarbiturates is generally recommended. The histamine-releasing opioids, such as morphine and meperidine, theoretically should also be avoided. Should opioid premedication be required, fentanyl is suggested. Finally, the use of H_2 receptor blockers (cimetidine, ranitidine) may provoke bronchospasm. Histamine mediates bronchoconstriction by H_1 receptors, whereas bronchodilation is mediated by H_2 receptors; hence, selective blockade of H_2 receptors could unmask unopposed bronchoconstriction.

Although the routine use of antisialogic agents as premedication has all but disappeared, these drugs may be useful in the asthmatic patient by decreasing the cyclic GMP release that mediates bronchospasm. Additionally, they dry up airway secretions that can provoke bronchospasm. As many find the anticholinergic effects of flushing, dry mouth, and tachycardia distressing, it is probably better to avoid premedication with the anticholinergics and include them as part of the induction of general anesthesia.

Myasthenia Gravis

Myasthenia gravis (MG) is an autoimmune disease of neuromuscular transmission. Thymomas are relatively frequent in patients with MG, and thymectomy has been shown to improve the patient's outcome.[44] A scoring system that has a predictive accuracy of 90% has been devised to determine which patients will require postoperative ventilatory support.[29] A score of $\geq$ 10 points using the following criteria means that ventilatory support will be required:

Duration of MG $\geq$ 6 years	12 points
Other respiratory disease	10 points
Pyridostigmine $>$ 750 mg/day	8 points
Vital capacity $\leq$ 2.9 L	4 points

Acute Respiratory Disease

Because upper respiratory infections are common, especially in children, the anesthesiologist frequently is required to decide whether to proceed with the planned operation or cancel it. Although earlier the practice was often to wait until all evidence of infection had cleared, more recent studies suggest that anesthetic administration to the patient with mild upper respiratory disease does not increase the risk of postoperative pulmonary complications.[35, 45, 59] (See also the article by Daran Haber elsewhere in this issue.)

Spinal Cord Injury

Injuries of the cervical and high thoracic segments of the spinal cord are associated with varying degrees of respiratory failure.

During the first 24 to 48 hours after injury, the patient may require surgical intervention for reduction of extremity fractures, stabilization of unstable vertebral fractures, or intrathoracic, intra-abdominal, or intracranial procedures. However, if at all possible, emergency surgery planned during the first 24 to 48 hours after injury should be delayed to maximize ventilatory reserve. Measures should include intermittent positive pressure ventilation, chest percussion, postural drainage, and assisted cough. The presence of pre-existing lung disease should be established and treated.

Lesions at C2 to C4 generally result in complete apnea and the patient is dead on admission. Partial lesions at this level produce sleep apnea. These patients generally hypoventilate even when awake. They are at the greatest risk of developing sleep apnea during the first 5 nights after injury, most likely because of a defect in the medullary ventilatory center secondary to edema and ischemia from lower cord trauma that extends cephalad.

Lesions at the level of C4 to C5 result in major loss of diaphragmatic movement and gross impairment of ventilation. C5 to C6 lesions are accompanied by reduced ventilation secondary to partial loss of diaphragmatic movement.

If the lowest uninvolved segment is at or below C6, diaphragmatic innervation is intact, but ventilation is reduced because of intercostal paralysis. This may reduce the tidal volume by 60% if rib cage expansion is abolished. In addition, vital capacity may initially be only 30% of the predicted value. Because of decreased vital capacity, inspiratory capacity, and expiratory reserve volume, the patient is unable to cough and clear secretions from the upper respiratory tract. In addition, the acute spinal cord injury patient may have aspirated the stomach contents, and a ventilation-perfusion mismatch may exist.

Evaluation of pulmonary function includes a chest radiograph to exclude rib fracture, pneumothorax, atelectasis, and aspiration. Pa_{CO_2} is a reliable dictator of alveolar ventilation; Pa_{O_2} reflects the degree of $\dot{V}/\dot{Q}$ mismatch. Atelectasis and pulmonary vasoconstriction secondary to catecholamine release is the most likely explanation for the development of $\dot{V}/\dot{Q}$ mismatch.

Improved vital capacity and forced expiratory volume guides further respiratory support and evaluates recovery of respiratory function. Factors contributing to improved pulmonary function include decreased cord edema, decreased use of respiratory depressant drugs, improved diaphragmatic function and strength, and strengthened accessory muscles of respiration.

TYPE OF SURGERY

The site and reason for surgery is of major importance in formulating an anesthetic plan to reduce risk in the patient with respiratory diseases. Several specific situations can pose special problems.

Abdominal Surgery

Upper abdominal surgery causes major changes in pulmonary function and mechanics. Postoperative these patients have a superimposed restrictive ventilatory defect, characterized by a reduction in the FVC, which may be severe on the first postoperative day and gradually improves during the next 72 hours. Eight per cent of pulmonary function is recovered by the third postoperative day.[4] Consistent with the restricted ventilatory defect, measured lung volumes, especially the FRC, are reduced. Although pain occurs on deep inspiration the reduction in lung volumes cannot be explained on this basis alone. The marked reduction in FRC with no visible changes on the chest film has resulted in the use of the term microatelectasis to designate the hypoxemic postoperative patient. As the term implies, with reduction in lung volume, physiologic right-to-left shunting increases. Respiratory rate increases, tidal volume decreases, and the tendency for the patient to sigh or take a deep breath is reduced. Even with pain relief, total reversal of this breathing pattern does not occur. Thus, the patient who has a significant restrictive defect or established obstructive lung disease is at high risk when an upper abdominal operation is performed. There is a superimposed restrictive defect, however transient, that may make it impossible for patients to maintain ventilation and oxygenation. For this reason most postoperative maneuvers are designed to increase lung volume and reestablish a nearly normal functional FRC.

Thoracic Surgery

The patient with respiratory disease who is having thoracic surgery usually has a lung tumor. Approximately 90% of solitary pulmonary nodules are malignant. The remaining 10% of tumors that are benign are mainly hematomas (80%), angiomas, lipomas, leiomyomas, fibromas, and chondromas. The most common type of lung cancer is squamous cell carcinoma (60%). It is generally slow growing and slow to metastasize. Large cell and small cell carcinomas each account for 15% of malignant tumors and tend to grow rapidly and metastasize early. Adenocarcinomas, the majority of the remaining tumors, are usually small peripheral lesions with moderate growth rates.

Almost all lung tumors with the possible exception of small-cell tumors that metastasize very rapidly are considered for resection.

Preanesthetic Evaluation. Standard evaluation of the patient with suspected lung cancer is outlined in Table 3. Assessment before lung resection should indicate the patient's ability to survive the planned procedure. Most patients with poor outcomes succumb to respiratory failure or heart disease.

All thoracic surgery patients should have pulmonary testing both before and after inhalation of bronchodilators as well as arterial blood gas analyses. Several studies have identified patients at risk for postoperative complications after pulmonectomy (Table 4).

The criteria for performing a pneumonectomy with minimum

Table 3. *Evaluation of the Patient with Suspected Pulmonary Tumor*

SYMPTOMS AND/OR CHEST X-RAY SUSPICIOUS FOR LUNG CANCER*	
Evidence of Metastases	*No Evidence of Metastases (tissue diagnosis done through)*
1. Scans of liver, bone, brain	1. Bronchoscopy
2. Consider biopsy of liver, bone brain	2. Mediastinoscopy
3. If search for another primary site is indicated, do IVP, BE, UGI	3. Needle biopsy 4. Mediastinotomy
4. Invasive diagnostic studies as indicated	5. Scalene node biopsy

* History and physical examination included CBC, SMA-18, ECK, chest x-ray, urinalysis, purified protein derivative tuberculin test and anergy panel, sputum for culture and sensitivity and cytology, hilar oblique tomograms, thoracic CT scan, and pulmonary function tests.

ICP = intravenous pyelogram, BE = barium enema, UGI = upper gastrointestinal series.

Adapted from Daitch, JS: The patient with suspected pulmonary lobectomy. Anesthesiology News 13:12, 1987; with permission.

Table 4. *Analyses of the Results of Lung Function Tests Can Be Used to Predict Outcome of Pulmonary Surgery*

STUDY	CRITERIA	OUTCOME
Gaensler et al[19] (1955)	MVV < 50% of predicted FVC < 70% of predicted	Mortality 40%
Mittman[38] (1961)	MVV < 50% RV/TLC > 50% of predicted "nonspecific abnormalities" on EKG	Mortality 46%
Boushy et al[6] (1971)	$FEV_1 < 2$ L $FEV_1/FVC < 50\%$ Age > 60 years	Mortality 40%
Lockwood[30] (1973)	VC < 1.85 L FVC < 1.70 L RV > 3.30 L TLC > 7.90 L RV/TLC > 47% $FEV_1 < 1.2$ MVV < 28 c/net	Very high risk
Boysen et al[7]	MVV < 50%	1 year survival 61%
Olsen et al[43] (1978)	$FEV_1 < 2$ L	
Miller et al[36] (1981)	MVV > 55% $FEV_1 > 2$ L	Predicted overall mortality 0.2%, after pneumonectomy 4.4%

MVV = maximum voluntary ventilation, FVC = forced vital capacity, RV = residual volume, TLC = total lung capacity, FEV_1 = forced expiratory volume in 1 second, VC = vital capacity.

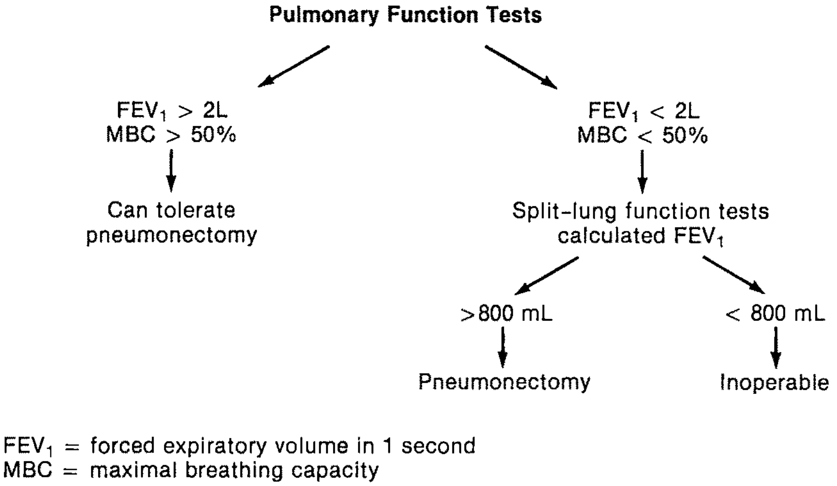
Pulmonary Function Tests
$FEV_1 > 2L$
$MBC > 50\%$
Can tolerate pneumonectomy
$FEV_1 < 2L$
$MBC < 50\%$
Split-lung function tests calculated FEV_1
>800 mL
Pneumonectomy
< 800 mL
Inoperable
FEV_1 = forced expiratory volume in 1 second
MBC = maximal breathing capacity

Figure 2. Operability criteria for pneumonectomy. FEV_1 = forced expiratory volume in 1 second, MBC = maximal breathing capacity.

morbidity and mortality are outlined in Figure 2. The criteria to ensure optimal results with a lobectomy, however, are less well known. Because less of the lung is resected, less preoperative pulmonary reserve is needed than for a pneumonectomy. The minimum criteria for lobectomy include (1) maximal breathing capacity (MBC) >40% of predicted value, (2) $FEV_1 > 1$ L, and (3) $FEV_{25\%-75\%} > 0.6$ L.

Patients who do not reach these minimum criteria may still be surgery candidates depending on the results of split-lung function testing. Split-lung ventilation and perfusion studies help determine the calculated postoperative pulmonary reserve. A calculated residual $FEV_1 > 800$ mL is compatible with a reasonable ventilatory reserve, such that a patient can function normally.[43] Also, hypercarbia develops when FEV_1 is < 800 mL.

This calculated lower limit of 800 mL for postoperative FEV_1 is advocated because almost all patients who had lung resection with a $Paco_2$ greater than 45 mm Hg died postoperatively of respiratory failure.[55, 56] Thus, patients scheduled for pneumonectomy with an FEV_1 of 2 L or for lobectomy with an FEV_1 of 1 L are evaluated with split-lung function tests to ensure that a postresection FEV_1 greater than 800 mL is possible.

When such criteria are used to determine operability, mortality is significantly reduced. Nevertheless, even when these minimum criteria are not reached, thoracic procedures may still be performed for emergent or life-threatening conditions, and mortality rates are less than 35%.[22]

Cardiovascular assessment is also important because tobacco smoking, often the cause of the tumor, can affect all body systems. Cardiopulmonary risk for lung resection is summarized in Table 5.[1]

Table 5. *Lung Resection and Cardiopulmonary Risk*

	VERY LOW RISK	MODERATE RISK	VERY HIGH RISK
Cardiac	Normal cardiac size and function Normal blood pressure Normal ECG	CAD with angina, MI, or ECG changes Cardiac dysrhythmias Systemic hypertension Valvular heart disease	Intractable CHF ventricular dysrhythmias Uncontrolled MI
Pulmonary	Normal arterial blood gases Satisfactory mechanical lung function ($FEV_1 >$ 70%)	Hypoxia with a normal $PaCO_2$ Reduced pulmonary function (FEV_1 of 35%–70%)	Poor mechanical function (FEV_1 < 35%) Irreversible CO_2 retention ($PaCO_2 > 45$ mm Hg)

Adapted from Ali MK, Ewer MS: Preoperative cardiopulmonary evaluation of patients undergoing surgery for lung cancer. Cancer Bull 32: 100–104, 1980; with permission.

CONCLUSIONS

The most important factors in the preanesthetic assessment of the patient with respiratory disease include careful history taking, physical examination, chest radiograph, arterial blood gas analyses, and spirometry. The extent of disease is a predictor of outcome. Although tobacco smoking contributes to the severity of the pathologic condition, sudden cessation does little to improve outcome. In addition, an acute respiratory infection should not automatically mean cancellation of surgery.

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Preanesthetic Assessment of the Patient with Renal Disease

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The kidneys play a key role in maintaining body homeostasis, as they are responsible for regulating the water, acid-base, and electrolyte balances. They eliminate nitrogenous wastes produced during protein metabolism, synthesize hormones involved in blood pressure regulation and red blood cell production, and are the site of excretion of many commonly used drugs.

In 1985, it was estimated that there were more than 72,000 patients in the United States maintained on some form of dialysis.[12] These patients require surgical intervention much more frequently than does the general population. For example, Hata and associates[17] reported that 28% of their patients on home dialysis required major surgical intervention during a 3-year study period, and Pinson and colleagues[28] found that their chronic dialysis patients averaged 1.1 access and 0.28 nonaccess procedures per year. Regardless of the type of surgical procedure required, patients with acute renal failure (ARF) or chronic renal failure (CRF) have a decreased capacity to respond to perioperative stress, and unless they are evaluated and managed appropriately, high perioperative morbidity and mortality will result.

DETECTION OF RENAL DYSFUNCTION DURING ROUTINE PREOPERATIVE EVALUATION

Every patient scheduled to receive a regional or general anesthetic does not require blood tests or timed urinary collection to assess the glomerular filtration rate (GFR). Patients younger than the age of

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40 years do not routinely have preoperative measurements of blood urea nitrogen (BUN) or creatinine. If these younger patients have asymptomatic but clinically significant renal disease, the risk of an adverse response to surgery and anesthesia is increased.

A carefully obtained history and a urinalysis will help the anesthesiologist select those patients who require additional laboratory testing for assessment of renal function. Abnormalities noted on physical examination (with the exception of hypertension) occur very late in the course of CRF, and therefore a normal physical examination does not exclude the presence of significant renal dysfunction. Historical clues to the possible presence of renal disease include (1) a history of diseases that commonly affect the kidney such as diabetes mellitus, systemic lupus erythrematosus, peripheral vascular disease, and hypertension; (2) a history of exposure to drugs that are nephrotoxic such as aminoglycoside antibiotics, chemotherapeutic agents, and anti-inflammatory agents; (3) a history of abnormal urinary sediment, which may be secondary to chronic glomerulonephritis, chronic pyelonephritis, or other intrinsic kidney diseases; (4) a family history of inherited kidney disease such as polycystic kidney disease; and (5) a history of urologic problems such as benign prostatic hypertrophy.

Urinalysis is useful as a routine screening test, as it is inexpensive, readily available, and informative. The presence of glucosuria usually indicates that the blood sugar is elevated and may indicate that the patient is diabetic with or without diabetic nephropathy. A protein concentration of >10 mg/dL on the "spot" urine suggests that significant proteinuria is present and thus warrants further investigation. Most commonly, proteinuria is secondary to increased protein filtration by diseased glomeruli. The anesthesiologist should consider the possibility that the loss of urinary protein may have created systemic hypoproteinemia, which will have significant implications for fluid management and choice of anesthetic agents that are highly protein-bound.

The specific gravity provides an estimate of the ability of the kidneys to concentrate and dilute the urine, but this value will be influenced by the amount of fluid ingested in the hours prior to obtaining the urine specimen. The pH is difficult to interpret without simultaneous data on the systemic acid-base balance.

Evaluation of the urinary sediment for the presence of heme, cells, and casts is useful in establishing a diagnosis of renal disease, but it is less useful for determining the functional capacity of the kidneys.

STAGES OF CRF

When assessing the perioperative risk for patients with CRF, the type of renal disease present is usually of less importance than the level to which the GFR has decreased.[33] (Fig. 1). Decreased renal reserve exists when the GFR is reduced but is still greater than 50% of the normal rate of 120 mL/min. Physiologic and biochemical ab-

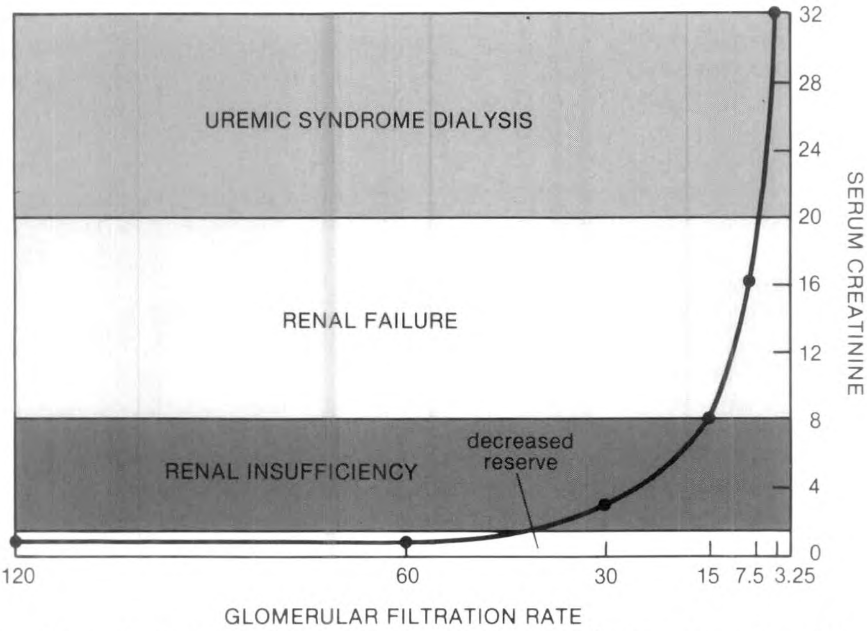

Figure 1. Illustration of the four stages of loss of renal function. (*From* Weir PHC, Chung FF: Anaesthesia and chronic renal failure. Can Anaesth Soc J 31:469, 1984; with permission).

normalities are absent, and the anesthetic goal is to maintain renal perfusion to minimize the risk of postoperative deterioration in renal function.

When the GFR is between 25 and 50 mL/min renal insufficiency is present. At this stage hypertension is often present and anemia may appear as well, but other systemic manifestations of uremia are lacking. With careful attention to fluid and electrolyte balance, perioperative morbidity should not be greatly increased.

When the GFR falls to between 10 and 25 mL/min, there is frank renal failure, and when it falls to below 10 mL/min, uremia is present. These two groups of patients are usually entered into a dialysis program to decrease morbidity and mortality of CRF.

When a patient with severe renal failure requires surgery, the anesthesiologist must thoroughly evaluate the patient preoperatively and look for manifestations of CRF that can influence perioperative risk and factors that might respond to preoperative treatment.

PHYSIOLOGIC ABNORMALITIES IN PATIENTS WITH CRF

Fluid Balance

Dialysis-dependent patients with CRF easily can become fluid overloaded during the perioperative period and develop intraoperative or postoperative pulmonary edema. The margin of safety between insufficient and excessive fluid administration is markedly decreased in those with CRF. Although evidence of pulmonary congestion on the physical examination and chest radiograph may be secondary to fluid overload alone, congestive heart failure also may contribute to the clinical picture. Congestive heart failure may occur in patients with CRF secondary to the sustained effects of hypertension such as hypertensive cardiac disease or may be caused by ischemia. Cardiac failure may be exacerbated by the presence of an arteriovenous (AV) fistula or a shunt used for hemodialysis.

Because of an inability to regulate extracellular fluid volume, patients with CRF may not tolerate the perioperative administration of blood, colloids, or crystalloids. Intraoperative management of dialysis-dependent patients with CRF is simplified if the patients are hemodialyzed 12 to 24 hours before surgery. Dialysis removes excess extracellular fluid and can bring the patients down to their "dry" weight. Central hemodynamic monitoring with either a central venous or pulmonary artery catheter is helpful when major blood loss or fluid shifts are anticipated during surgery. These monitors can help the anesthesiologist avoid excessive fluid infusion while at the same time prevent excessive dehydration and its potentially adverse effects on intraoperative renal blood flow.

Approximately 10% of patients with CRF are unable to conserve salt and water in a normal fashion and therefore are at risk of developing hypovolemia unless a high salt and water intake is maintained. These patients are usually normotensive or mildly hypotensive and do not have peripheral edema. Fluid and salt restriction in these patients is potentially dangerous and may result in a further (prerenal) deterioration in renal function.

Electrolyte Abnormalities

Hyponatremia. Patients with CRF have a decreased ability to conserve or excrete free water. Hyponatremia and hypernatremia are uncommon in these patients because water intake is adjusted by the thirst mechanism. Hyponatremia, when present, usually results from water retention in excess of sodium retention. Because total body sodium is increased, it is more appropriate to correct the hyponatremia by restriction of sodium and water intake than it is to administer additional sodium.

Hyperkalemia. Hyperkalemia is the most serious electrolyte abnormality in patients with CRF. Conduction defects and ventricular dysrhythmias may result, including ventricular tachycardia and ventricular fibrillation. Hyperkalemic cardiac arrest may be the leading cause of intraoperative mortality in patients with renal failure who have been prepared poorly for anesthesia and surgery.

Electrocardiographic changes are usually present when the serum potassium level exceeds 7 mEq/L. The earliest changes are the development of tall peaked T waves. As the serum potassium continues to increase, the QRS complex duration becomes prolonged, the P wave loses amplitude and eventually disappears, and the QRS complex and T wave merge to produce the classic sine wave of hyperkalemia.

It has been recommended that elective surgery be postponed if the serum potassium exceeds 5.5 mEq/L.[33] Hampers and associates[16] evaluated morbidity and mortality in patients with CRF undergoing major surgery. Forty-four procedures were described in 33 patients. In only two instances was the preoperative potassium greater than 6 mEq/L. One of these patients (with a preoperative potassium of 6.5 mEq/L) died of an intraoperative cardiac arrest; this was the only death in the series. Patients with severe renal dysfunction (acute or chronic) poorly handle potassium loads that would have insignificant effects in patients with normal renal function. Transfusion of old blood, extensive surgical tissue trauma, hemolysis, and rhabdomyolysis can all result in release of potassium that may precipitate life-threatening hyperkalemia either intraoperatively or in the early postoperative period. Brenowitz and colleagues[5] reported that 31% of patients with CRF required treatment for hyperkalemia in the first 24 postoperative hours after major surgery. Patients who received intraoperative blood transfusions developed postoperative hyperkalemia that required treatment 44% of the time, whereas patients who did not receive blood transfusions developed postoperative hyperkalemia requiring treatment only 25% of the time. In this series, hyperkalemia was the most frequent postoperative complication.

The development of a respiratory or metabolic acidosis also can produce a significant increase in the serum potassium even though the total body potassium remains unchanged. Arterial blood gas analyses should be obtained preoperatively. Most patients with severe renal failure demonstrate a metabolic acidosis with respiratory compensation ($Paco_2$ is decreased). If, after induction of anesthesia, hyperventilation is not maintained, the pH decreases and a rapid increase in the serum potassium may result. It has been proposed that this mechanism may account for some of the unexplained operative deaths reported in patients with renal failure.[14]

When emergency surgery is required in a uremic, hyperkalemic patient, a decision must be made whether there is enough time for peritoneal or hemodialysis prior to surgery. If surgery cannot be delayed for a few hours, emergency treatment for hyperkalemia should be initiated with sodium bicarbonate, 50% dextrose and insulin, and calcium infusion if electrocardiographic abnormalities of hyperkalemia are present (Table 1). Both the sodium bicarbonate and the 50% dextrose and insulin combination shift potassium from the extracellular to the intracellular space, whereas calcium protects the heart from the proarrhythmic effects of hyperkalemia. These dysrhythmias occur secondary to an imbalance in the ratio of potassium to calcium that adversely affects myocardial conduction. Calcium infusion restores the calcium to potassium ratio without influencing the serum potassium level.

Ion exchange resins such as Kay-exalate exchange sodium for potassium and decrease the total body stores of potassium. However,

Table 1. *Guidelines for the Emergency Treatment of Hyperkalemia*

TREATMENT	MECHANISM	ONSET AND DURATION OF EFFECT
Calcium chloride 1–2 g, IV slowly (Repeat 1–3 times every 10 min if necessary); monitor EKG	Antagonism of K^+ effects on the heart	Onset immediate, duration 10–15 min
Regular insulin 10–15 U IV plus 50 g D50 IV over 20 min	Shifts K^+ from extracellular to intracellular space	Onset in 5 min, duration 0.5–3 hr
Sodium bicarbonate 50–100 mEq IV slowly	Shifts K^+ from extracellular to intracellular space	Onset in minutes, duration 0.5–1 hr
Kayexalate 50 g (sodium polystyrene sulfonate) plus 200–500 mL 10% sorbitol as an enema	Removes K^+ from body via the GI in exchange for Na^+	Onset in 2–3 hr, duration 6–8 hr

when emergent surgery is required, use of these resins is impractical because of the time needed before a significant effect on serum potassium occurs.

Hyperphosphatemia and Hypocalcemia. Disorders of phosphate and calcium balance are interrelated in patients with CRF. Because of the lowered GFR present in patients with CRF, phosphate levels become elevated. In the presence of severe renal dysfunction, vitamin D synthesis is altered, thus producing a severe impairment of calcium absorption by the gut and hypocalcemia. The combination of a low serum calcium level and a high phosphate level stimulate the secretion of parathormone from the parathyroid glands. Under the influence of elevated parathormone levels, calcium is mobilized from the bones that may eventually contribute to the development of renal osteodystrophy.

Patients with renal osteodystrophy are prone to spontaneous fractures. The anesthesiologist must therefore position and pad these patients carefully.

Hypocalcemia is usually asymptomatic in patients with CRF. Tetany is unusual although the reason for this is uncertain. It has been postulated that metabolic acidosis raises the ionized calcium level without affecting the total serum calcium. It has also been suggested that acidosis directly decreases neuromuscular irritability and thereby modifies the effects of hypocalcemia. Tetany, however, may be precipitated by rapid or full correction of metabolic acidosis with bicarbonate. Hypocalcemia potentiates the toxic effects of hyperkalemia on the myocardium.

The usual treatment for hyperphosphatemia and hypocalcemia is the use of oral phosphate binders such as aluminum hydroxide. When the phosphate level decreases, the calcium level often increases. Dialysis can also be used to control these electrolyte abnormalities.

Hypermagnesemia. Hypermagnesemia is often present in patients with CRF. It infrequently produces symptoms in the dialysis-dependent patient unless the patient is abusing magnesium containing antacids or cathartics. If hypermagnesemia is present, prolongation of the effects of nondepolarizing muscle relaxants should be expected.[13]

CARDIOVASCULAR COMPLICATIONS IN PATIENTS WITH CRF

There are many pathologic changes in the cardiovascular system in patients with renal disease.

Hypertension

Hypertension is a common clinical finding in patients with CRF. In the majority of patients it is a result of fluid overload and therefore responds well to dialysis and restriction of salt and water intake. Less frequently, hypertension is secondary to renal vascular disease or other conditions associated with increased renin output by the kidneys. Dialysis is unlikely to control the blood pressure in these patients, and potent antihypertensive agents are usually necessary. Occasionally, nephrectomy is required to control hypertension.

Pericarditis

Cardiac enlargement or the presence of pleuritic chest pain that is modified by changes in patient position should arouse suspicion for the presence of uremic pericarditis. Reports in the 1960s and 1970s described cardiac tamponade in up to 6% of patients with CRF and in up to 55% of patients with uremic pericarditis.[2, 20] Uremic pericarditis with or without cardiac tamponade has become much less common with the widespread availability of dialysis. If uremic pericarditis does occur, it will often respond to aggressive dialysis therapy. Systemic anticoagulation is usually avoided during hemodialysis of patients with uremic pericarditis, as bleeding into the pericardial sac may occur and result in acute pericardial tamponade. If fluid accumulation progresses despite increased frequency of dialysis, additional interventions such as repeated pericardiocentesis, surgical creation of a pericardial window, or pericardectomy may be necessary.[20]

Accelerated Atherosclerosis. Patients with CRF on long-term hemodialysis have accelerated development of atherosclerosis.[21] Ischemic heart disease is commonly present in patients with CRF[1, 32] and may be a consequence of a systemic disease that produces the renal failure (e.g., diabetes mellitus) or CRF itself. Cardiac ischemia frequently is asymptomatic in patients with autonomic neuropathy (whether from diabetes or uremia). Goldman et al[15] demonstrated that a serum creatinine level greater than 3 mg/dL was an independent cardiac risk factor for patients undergoing noncardiac surgery. Because of the frequency of atherosclerotic cardiovascular disease in patients with CRF, it is essential that during preoperative evaluation a careful cardiac history be obtained and, if indicated, appropriate further cardiac evaluation and cardiac consultation be obtained prior to elective surgery.

ANEMIA IN PATIENTS WITH CRF

Anemia was once a universal finding in patients with advanced CRF. However, with the clinical introduction of recombinant human erythropoietin therapy, the hemoglobin level in uremic patients has become more variable.

The anemia is normochromic and normocytic, and multiple factors contribute to its development. Decreased erythropoietin synthesis plays a major role; other contributing factors are decreased red blood cell survival, bone marrow toxicity from uremia, iron deficiency, and deficiencies of folate and vitamin B_{12}, which develop because of the combination of poor intake and dialysis removal of these vitamins.[11]

When erythropoietin production is markedly decreased, red blood cells are still produced by the bone marrow, however, the hemoglobin level rarely increases above the range of 7 to 9 g/dL. Dialysis-dependent patients with CRF usually tolerate hematocrit levels in the range of 15% to 25% with minimal symptoms. Because the anemia develops slowly, compensatory mechanisms can be used to maintain tissue oxygen delivery at near normal levels. The blood volume and cardiac preload are usually normal or increased. Left ventricular afterload is decreased secondary to the effects of a low hematocrit level on blood viscosity and the presence of an AV fistula or shunt. These factors contribute to an increased cardiac output and tissue blood flow, which compensates for the anemia induced decrease in blood oxygen carrying capacity.[7] In addition, at the tissue level, oxygen dissociation from the blood is enhanced by a rightward shift in the oxyhemoglobin dissociation curve that is caused by the combined effects of metabolic acidosis and increased concentration of 2,3 diphosphoglycerate.

It is essential for the anesthesiologist to understand that the anemic patient with CRF is dependent upon a hyperdynamic circulation to maintain tissue oxygenation. Efforts should be made to choose an anesthetic approach that will interfere minimally with cardiac output.

Preoperative blood transfusion usually is not indicated for the well-controlled uremic patient with a hematocrit level above 20%, even for major surgery. If the hematocrit level is less than 20% a transfusion should be administered slowly to minimize the risk of pulmonary edema. Increasing the hematocrit level to 25% is usually adequate. Ideally, leukocyte-poor packed red blood cells should be given during a dialysis treatment to remove excess potassium and fluid. Transfusions also should be considered for patients with CRF with symptoms believed to be secondary to anemia, even if the hematocrit level is above 20%.

Blood should not be withheld from patients with CRF for fear that it will make rejection of a subsequent renal transplant more likely. Transplant recipients who have received pretransplantation blood transfusions actually have an improved allograft survival for unknown reasons. Recombinant human erythropoietin currently is used to treat the anemia of CRF.[8] Patients who have improved their hematocrit level with this treatment normalize cardiac function.[23] Treated patients have increased problems with hypertension and thrombosis of AV fistulas.[8] Because the cardiovascular status of patients receiving this hormone therapy differs from most patients with CRF, it is important to obtain a history of use of this drug.

Coagulopathy

The presence of a coagulopathy should be suspected in all patients with CRF. Most frequently, coagulation abnormalities are secondary to a qualitative defect in platelet function that results in decreased platelet aggregation. The platelet number is usually normal or mildly decreased. Increased plasma levels of guanidinosuccinic acid cause a decline in the availability of platelet factor 3, subsequently inhibiting secondary platelet aggregation.[18] However, removal of this toxin by dialysis only partially corrects the abnormal hemostasis of uremia, thus suggesting that other factors must be involved.[22] Several other factors may be involved including a functional defect in cyclooxygenase that leads to reduced thromboxane formation, enhanced PGI2 formation by vessel endothelial cells, increased plasma levels of parathyroid hormone, and altered components of circulating factor VIII. Anemia may also affect coagulation by altering the rheologic component of the platelet-vessel wall interaction.[22]

Patients receiving hemodialysis usually are systemically heparinized. Effects of heparin are evident for several hours after completion of dialysis unless protamine has been infused. Other patients with CRF may be maintained on warfarin or antiplatelet drugs in an attempt to prevent thrombosis of their AV shunt or fistula.

Evaluation of the coagulation status of patients with CRF includes eliciting a history of abnormal brusing or bleeding, use of warfarin or antiplatelet drugs, and noting the time of the last dialysis treatment. Minimal laboratory testing should include measurement of the prothrombin time (PT), partial thromboplastin time (PTT), platelet count, and template bleeding time. The template bleeding time is the best single test that correlates with the risk of clinical bleeding in the patient with CRF. Regional anesthesia should be avoided unless the coagulation status of the patient is known.

Although hemodialysis often results in an improvement in platelet function, the improvement is variable and therefore unpredictable. Cryoprecipitate infusion improves the bleeding time presumably through effects on factor VIII: von Willebrand factor complex (FVIII:vWF).[19] Nevertheless, there is a substantial risk of transmitting hepatitis with this blood product. 1-Deamino-8-D-arginine vasopressin (DDAVP) is currently the most appropriate therapy for correction of prolonged bleeding times in patients with CRF who require surgery. DDAVP releases FVIII:vWF from storage sites into the plasma, producing a two to four fold elevation of the plasma level. In one controlled trial involving 12 patients with very prolonged bleeding times, there was a significant shortening of the bleeding time after DDAVP.[24] A dose of 0.3 μg/kg should be given intravenously over 20 minutes during close blood pressure monitoring. The peak effect (shortening of bleeding time) occurs after 1 to 2 hours and the duration of effect is from 6 to 12 hours. Repeated doses are less effective presumably because of depletion of cellular stores of FVIII:vWF.

NEUROLOGIC COMPLICATIONS IN PATIENTS WITH CRF

Uremic Encephalopathy

The degree of encephalopathy in patients with severe CRF depends upon the rapidity of progression of uremia; the more rapid the progression, the more severe the mental status changes. Manifestations of uremic encephalopathy vary from subtle personality changes to lethargy, seizures, and coma.[31] When an acute change in mental status develops in a patient with CRF, other causes should be considered, including subdural hematoma, cerebral vascular disease, and central nervous system (CNS) infections. Uremic encephalopathy improves with dialysis therapy.[29, 30]

Peripheral and Autonomic Neuropathy

Peripheral neuropathy occurs frequently in uremic patients. Clinical or electrical evidence of neuropathy was found in 50% of patients with CRF in one large series.[27] Characteristically, a distal neuropathy involves the legs more often than the arms.[29, 30] It is advisable to avoid using succinylcholine, as cases of hyperkalemic response have been reported.[32a] Anesthesiologists may also choose to avoid the use of regional anesthesia in the involved limbs because of potential medical-legal implications, although careful documentation of the presence of uremic neuropathy should minimize this risk.

Autonomic neuropathy should be suspected in patients with a history suggestive of orthostatic hypotension. For this reason blood pressure and pulse should be checked with the patient in the supine and sitting or upright positions during the preoperative physical examination. Autonomic neuropathy results in a generalized decrease in the sympathetic nervous system response to hypovolemia or anesthetic induced cardiac depression. A greater risk of intraoperative and postoperative hypotension is expected in these patients, and a lower threshold for placement of invasive hemodynamic monitors should be used.

UREMIC GASTROENTERITIS IN CRF

Nausea, vomiting, diarrhea, anorexia, and chronic hiccups are features of uremic gastroenteritis.[34] Of particular concern to the anesthesiologist are the delayed gastric emptying time, hyperacidity, and increased gastric juice volume that have been described in patients with CRF.[26] Full stomach considerations must therefore apply when the patient is brought to the operating room for general anesthesia and during emergence.

INFECTION IN CRF

Nosocomial and opportunistic infections are commonplace in CRF. Shunt infections are a major problem, and surgical removal of the shunt is sometimes necessary. Postoperative sepsis, which is often secondary to pneumonia, is a major contributor to morbidity and mortality in CRF, particularly when an emergent procedure has been performed.[4] Uremia appears to suppress leukocyte chemotaxis and immune response directly. This suppression along with the need to frequently place tubes and catheters for dialysis access contributes to the increased risk of infection. Elective surgery should be postponed if an active, untreated infection is present. Sepsis remains the most common cause of death in patients with uremia.

Because the incidence of hepatitis B infection is very high in patients on chronic hemodialysis, anesthesiologists should protect themselves from direct contact with blood and secretions.

ACIDOSIS IN CRF

When the GFR of patients with CRF falls below 20 to 30 mL/min, a metabolic acidosis with an increased anion gap develops secondary to accumulation of fixed acids (sulfates and phosphates) that are produced from normal metabolic processes. The rate and depth of respiration increase to compensate for the metabolic acidosis (Kussmaul's respiration), which helps keep the pH close to normal. Hyperventilation should be maintained after induction of anesthesia and into the recovery phase. Failure to do so can result in a significant decrease in pH and development of cardiac dysrhythmias from acute hyperkalemia.

Patients on chronic dialysis rarely have a pH less than 7.25 or a serum bicarbonate level less than 18 mEq/L. Sodium bicarbonate does not have to be given to these patients preoperatively, but the acid-base balance must be followed closely because the reserve to handle a superimposed acidosis (eg lactic acidosis) is significantly decreased. It has been suggested that the serum bicarbonate level should be 18 mEq/L or higher before surgery. Although treating the acidosis with sodium bicarbonate infusion is simple, serious side effects can occur and include production of acute hypokalemia and hypocalcemia. It is often stated that severe metabolic acidosis can depress the cardiovascular systems responsiveness to both endogenous and exogenous catecholamines.[9] This rationale often provides a basis for giving bi-

Table 2. *Effects of Dialysis on Uremic Abnormalities*

- Abnormalities that are corrected or improved by dialysis
  - Volume overload
  - Hyperkalemia, hyponatremia, hyperphosphatemia, hypocalcemia
  - Hypertension (if secondary to volume overload)
  - Congestive heart failure
  - Uremic pericarditis
  - Uremic encephalopathy
  - Peripheral neuropathy
  - Platelet dysfunction (variable response)
  - Metabolic acidosis
- Abnormalities that persist despite dialysis
  - Anemia
- Abnormalities that develop or are exacerbated by dialysis
  - Atherosclerotic cardiovascular disease
  - Hepatitis
  - Dialysis dementia
  - Dysequilibrium syndrome

carbonate to patients with all causes of metabolic acidosis. Although animal studies have supported this approach, the only available prospective clinical trial clearly demonstrated no beneficial effect on cardiovascular function when bicarbonate was used to correct severe metabolic acidosis associated with shock.[9] Despite this provocative study, it is still generally recommended that the pH be increased before surgery to greater than 7.25 and the bicarbonate level increased to greater than 18 mEq/L by administration of oral or intravenous bicarbonate.[3] The acidosis also can be corrected during dialysis. Table 2 summarizes the effects of dialysis in uremic patients.

SURGICAL RISK IN PATIENTS WITH CRF

The risk of surgery in the patient with CRF has been evaluated in several studies. The mortality rate in elective surgery has been consistently low, ranging from 0% to 4% for major surgery[5, 16, 28] to as high as 6% for major abdominal surgery.[4] Much more limited data are available regarding mortality rates when emergent surgery is required and optimal preoperative management is precluded. Borlase and associates[4] reported a 47% mortality rate (7/15) in hemodialysis patients with CRF who required emergency abdominal surgery. The authors noted that 5 of the 7 emergency surgery patients who died could have had the surgery earlier, on an elective basis. This approach might have resulted in a lower overall mortality rate.

Although mortality rates for elective surgery in dialysis-dependent patients with CRF are low, morbidity is substantial. Brenowitz and associates[5] reported that at least one postoperative complication developed in 61% of patients after major surgery. Hyperkalemia requiring medical treatment or emergency dialysis developed after 31%

of the operations, thrombosis of AV fistulas occurred after 22% of procedures, and pneumonia developed after 20% of the procedures, even though 88% of patients were dialyzed within 24 hours of surgery and only one had an elevated preoperative potassium level.

Similar results were reported by Pinson et al[28] (i.e., postoperative complication rate of 64% after 312 surgical procedures). Hyperkalemia was again the most frequent complication; it developed in 19% of patients. Factors that appeared to be related to the development of postoperative hyperkalemia included massive blood transfusion and the presence of an elevated preoperative potassium level. If it is obvious before the operation that a patient will require transfusions, it is preferable that the patient be given the transfusion during preoperative dialysis so that excess potassium and volume can be removed.

These studies demonstrate that despite the use of preoperative dialysis, a substantial complication rate can be expected when patients with CRF have major elective surgery.

The frequent presence of associated diseases such as hypertension, angina, congestive heart failure, and diabetes in patients with CRF contributes to high complication rates. These patients must be monitored closely after the operation for development of hyperkalemia, sepsis, bleeding, AV fistula thrombosis, fluid overload, myocardial ischemia, and hypotension. An intensive care unit is an appropriate location to care for the patient with CRF after major surgery.

PREOPERATIVE EVALUATION OF THE PATIENT WITH CRF

History

The etiology of the CRF should be noted. If it is secondary to a systemic disease such as diabetes mellitus or hypertension, other manifestations and complications of the disease should be sought. An estimate of the daily urine output should be made by either chart review or patient estimate. The patient with a salt losing nephropathy and high urine output is less likely to be fluid overloaded and will be more prone to develop intraoperative and postoperative hypovolemia. If the patient is maintained on a chronic dialysis program, the type (hemodialysis, chronic ambulatory peritoneal dialysis) and frequency of treatments should be recorded. It is important to record the time of the last presurgical hemodialysis because anticoagulation from heparin persists for a few hours. The patient should be asked if he or she has had any manifestations of uremia (see prior discussion) or complications of the disease and its treatment. The past surgical history and untoward reactions to anesthesia and medications should be recorded. Toxic effects may be present from drugs primarily metabolized by the kidney (e.g., digitalis).

Physical Examination

Except for the presence of hypertension, abnormalities on physical examination are absent until severe decreases in the GFR have

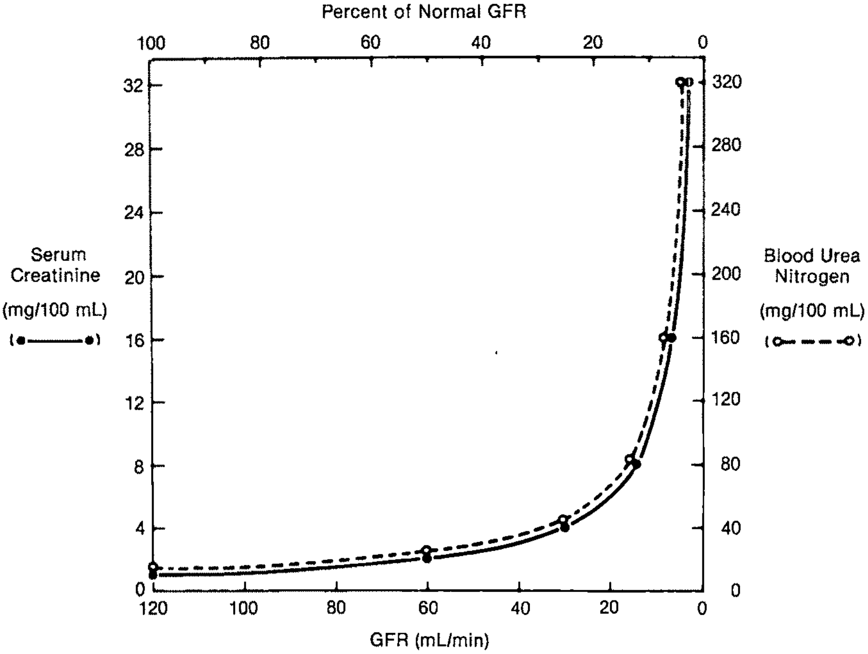

Figure 2. Relationship between BUN and creatinine versus glomerular filtration rate. (Reprinted by permission of *The New England Journal of Medicine, From* Kassirer JP: Clinical evaluation of kidney function-glomerular function. 285:385, 1971.)

occurred (e.g., GFR < 20 mL/min). These patients usually receive some from of chronic dialysis therapy, and the type and severity of abnormalities detected on preoperative physical examination are influenced by timing since the last dialyses.

A comprehensive preoperative examination is essential. Special attention should be paid to (1) measurement of the blood pressure and pulse to document the presence of hypertension and to evaluate whether orthostatic changes occur, (2) the patient's mental status to detect encephalopathy, (3) pulmonary system to detect râles and evidence of pleural effusion, (4) cardiac system to evaluate evidence of failure and/or pericardial effusion, and (5) neurologic system to detect a peripheral and/or autonomic neuropathy and asterixis. The anesthesiologist also should examine the location and function of existing AV fistulas or shunts and look for evidence of coagulopathy (bruises, petechiae).

Laboratory Evaluation

BUN and creatinine clearance are not very sensitive tests of renal function; the GFR has to be decreased by as much as 50% before the BUN and creatinine increase above the upper limits of normal (Fig. 2). In general, the GFR has a predictable inverse relationship with both the BUN and creatinine. For every 50% decrease in GFR, the BUN and creatinine double.

Creatinine is the end product of creatine metabolism. The daily production and release of creatine in an individual patient is fairly constant, and the amount released is directly related to the muscle mass of the patient. Therefore, cachectic individuals with normal GFR have a low serum creatinine level. Even if the GFR is decreased to 50% of normal, the creatinine level may remain within normal limits. The presence of a normal creatinine level may mislead the anesthesiologist, and if normal dosages of drugs are given that are primarily metabolized by the kidneys, toxic or prolonged effects may be seen. The creatinine level increases independent of GFR in muscular patients. Small increases in creatinine also have been reported in patients receiving cimetadine, which blocks renal tubular secretion of creatinine without affecting GFR.

The BUN also can be affected by factors other than a change in the GFR. Urea is produced in the liver as the main product of nitrogen metabolism. In patients with cirrhosis or other forms of end-stage liver disease, there may be inadequate liver function to produce a normal amount of urea for a given protein load. Under these conditions GFR is overestimated if it is based on the BUN. Patients who are overhydrated and those with low protein intake also have a low BUN and overestimation of GFR may result.

In contrast, the BUN may be increased for reasons other than a drop in the GFR. Anything that increases the protein load to the liver (high protein diet, gastrointestinal bleeding, hypermetabolic states with catabolism of muscle proteins) can result in an increased BUN without a change in GFR. Urea and creatinine are both freely filterable across the glomerulus, but urea, in contrast to creatinine, is partially reabsorbed by renal tubule cells. The fractional reabsorption of urea is increased at low urine flow rates, a situation associated with congestive heart failure and dehydration. The BUN increases out of proportion to any change in creatinine creating a state of prerenal azotemia. Prerenal azotemia should be suspected when the BUN/creatinine ratio is greater than 20:1. This ratio should always be calculated during the preoperative evaluation as it serves as a marker of the presence of mild renal hypoperfusion that if left untreated can progress to overt renal failure. Because renal function often improves with therapy aimed at increasing cardiac output and renal blood flow, elective surgery should be postponed until the prerenal azotemia resolves with appropriate therapy.

It is unlikely that the anesthesiologist will require a 24-hour urine collection for determination of the creatinine clearance to safely anesthetize a patient. However if the data are available, they should be used as a more accurate estimate of GFR than can be obtained from measurement of BUN or creatinine.

A complete blood count should be obtained to determine the baseline hemoglobin level and platelet count. Because a normal platelet count does not ensure normal platelet function, a bleeding time should be measured, and if it is significantly prolonged, the use of DDAVP should be considered.[24] An electrolyte panel should be evaluated and include determinations of sodium, potassium, and total CO_2. If possible, a potassium measurement should be available from within 4 hours of the patient's arrival in the operating room. It is also important to have a serum calcium (ionized), phosphate and magnesium level, if time permits. Arterial blood gas analyses should be obtained before induction of anesthesia not only to assess the patient's acid-base status but also to determine whether oxygenation is normal. It also helps establish the level $PaCO_2$ should be brought to intraoperatively.

A chest roentgenogram should be obtained to evaluate heart size (cardiomegaly may reflect hypertensive cardiovascular disease or pericardial effusion), pleural effusions, and pulmonary edema, (which can be caused by fluid overload, congestive heart failure, or uremic pneumonitis.

An electrocardiogram (ECG) should be obtained to diagnose ischemic heart disease, hypertensive cardiovascular disease, and pericardial effusion (low voltage). If severe hyperkalemia (7 mEq/L or greater) is present, an EKG should be obtained immediately to assess whether toxic effects of hyperkalemia on cardiac conduction are present. If hyperkalemic changes are detected immediate treatment is indicated (see Table 1).

PREOPERATIVE EVALUATION OF THE PATIENT WITH ARF

ARF is characterized by an abrupt decline in renal function associated with retention of nitrogenous waste products and subsequent elevation of the BUN and serum creatinine levels. The urine output is variable in ARF as oliguric and nonoliguric varieties have been well described. It has been estimated that between 30 and 60 patients per one million people receive hemodialysis for treatment of ARF. The need for surgical intervention in patients with ARF is not rare. Patients may require creation of an AV fistula on an elective basis, emergency surgery for treatment of major upper gastrointestinal bleeding, or abdominal surgery for treatment of a source of sepsis. Elective surgery is usually deferred in patients with ARF until recovery has occurred or until the nephrologist indicates that further recovery is unlikely.

When ARF develops postoperatively a mortality rate of greater than 50% can be expected, although aggressive treatment might reduce it to less than 40%.[6] Many patients with ARF are critically ill and renal failure is part of multiorgan failure. In one study when patients with ARF developing after abdominal surgery required a reexploration, major complications developed in more than 90%.[25]

Preoperative evaluation of the patient with ARF does not differ substantially from that previously described for the patient with CRF. There are, however, a few differences that are clinically important.

Patients with ARF are often in a hypermetabolic state secondary to the disease process that produced the ARF (e.g., septic shock or traumatic injury). Muscle breakdown is increased and the rate at which the creatinine and BUN rise between dialysis treatments also is increased. Because of the catabolic state, these patients can develop hyperkalemia quickly and the potassium level must be checked immediately before surgery and on a frequent basis intraoperatively and postoperatively.

Anemia in the patient with ARF is not as well-tolerated as in patients with CRF. Compensatory mechanisms are not fully used (such as increased cardiac output). Therefore the hematocrit level should be maintained close to 32; which is the optimal hematocrit level for critically ill patients.[10]

Determining the intravascular volume status can be difficult. If the cause of ARF was septic or hypovolemic shock, the patient may have been aggressively fluid resuscitated and be hypervolemic. If the patient has a nonoliguric variety of ARF or is in the diuretic (recovery) phase of ARF, the patient may be hypovolemic. Central venous or pulmonary artery catheters may be necessary to determine intraoperative fluid requirements. The goal is to prevent a new hemodynamic insult to already damaged kidneys.

Metabolic acidosis in patients with ARF may be secondary to renal failure alone but other causes must be considered and excluded. In particular, tissue ischemia inducing lactic acidosis should be excluded.

SUMMARY

The patient with renal disease, whether acute or chronic, presents many challenges to the anesthesiologist. A high postoperative complication rate can be expected, although mortality rates are not increased substantially after elective surgery. Severe renal failure produces physiologic abnormalities that affect almost all organ systems. A thorough preoperative evaluation (history, physical examination, and appropriate laboratory testing) helps the anesthesiologist detect those abnormalities and then plan an appropriate anesthetic technique. In addition the preoperative evaluation will determine if the patient is in optimal condition for surgery or whether surgery should be postponed to improve the overall medical condition of the patient.

An understanding of the physiologic derangements that develop in both acute and chronic renal failure help the anesthesiologist manage these interesting and difficult disease processes.

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Preanesthetic Assessment of the Patient with Endocrine Disease

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Many endocrine abnormalities are found in patients who present for surgery. Frequently, these disease processes may have a negative impact on anesthetic management. An understanding of the pathophysiology involved and identification of potential adverse interactions will help the anesthesiologist deal with these patients.

Some of the more common endocrine abnormalities are reviewed herein.

THYROID GLAND

The major thyroid hormones are thyroxine (T_4), produced by the thyroid gland, and L-3,5,3′-triiodothyronine (T_3). The latter hormone is more potent and less protein bound and is produced inside (20%) and outside (80%) the thyroid gland. Each hormone influences cellular metabolic activity. The production of thyroid hormones is initiated by active absorption of iodine from the gastrointestinal tract where iodine is reduced to an iodide and released into the plasma. Circulating iodide is taken up and concentrated 500-fold by the thyroid gland. In the gland, the iodide is oxidized by a peroxidase reaction to form iodine and then it is bound to tyrosine to form either monoiodotyrosine or diiodotyrosine. These compounds are then coupled enzymatically to form T_4 or T_3. Both hormones are bound to thyroglobulin and stored as colloid within the gland. A proteolytic enzyme causes release of these hormones from the thyroglobulin into the circulation. Thyrotropin (TSH) is produced in the anterior pituitary gland, and its secretion is regulated by thyrotropin-releasing hormone (TRH), which is produced in the hypothalamus. TSH is responsible for maintaining the uptake of iodide and proteolytic release of the thyroid hormone.

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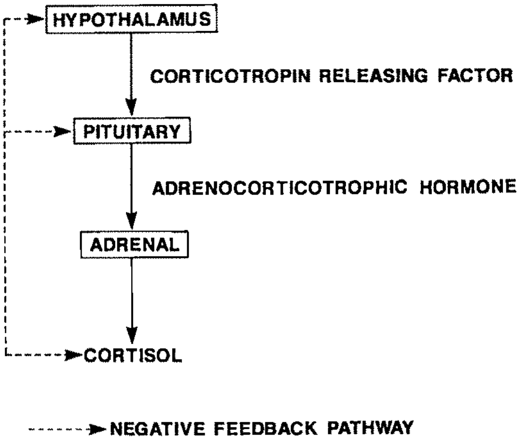
HYPOTHALAMUS
CORTICOTROPIN RELEASING FACTOR
PITUITARY
ADRENOCORTICOTROPHIC HORMONE
ADRENAL
CORTISOL
NEGATIVE FEEDBACK PATHWAY

Figure 1. Hypothalamic-pituitary axis.

A negative feedback loop dependent upon circulating levels of T_3 and T_4 exists to regulate both TSH and TRH. Excess iodine inhibits the synthesis and secretion of thyroid hormone[12] (Fig. 1).

The thyroid gland secretes 80 to 100 μg/day of T_4. The half-life of T_3 is 24 to 30 hours, whereas the half-life of T_4 is 6 to 7 days. The plasma normally contains 5 to 12 μg/dL of T_4 and 80 to 220 μg/dL of T_3. Thyroid hormone increases the intrinsic contractile state of cardiac muscle, increases the β-adrenergic receptor number, and decreases cardiac cholinergic receptors accounting for the cardiovascular changes from thyroid disease.

Diagnostic tests to ascertain thyroid status may appear bewildering to nonendocrinologists. A battery of standardized screening tests are available to confirm most thyroid-related diagnoses (Table 1). Total serum T_4 is the standard screening test for thyroid gland function. T_4 is elevated in hyperthyroidism 90% of the time and decreased in hypothyroidism 85% of the time. Because T_4 is influenced by thyroid hormone protein-binding capacity, an increase or decrease in thyroglobulin levels or protein binding will affect the total T_4 concentrations in euthyroid persons. Increased thyroglobulin levels have been seen in pregnancy, infants up to 6 weeks of age, (exogenous estrogens, methadone, heroin, clofibrate), infectious hepatitis, chronic liver disease, acute intermittent porphyria, hydatiform moles, and

Table 1. *Thyroid Function Tests*

	T_4	T_3RU	T_3	TSH
Normal values	1.5–5.0 $ng \cdot dL^{-1}$	25–35%	80–200 $ng \cdot dL^{-1}$	L8 $IU \cdot mL^{-1}$
Hyperthyroidism	↑	↑	↑	↔ ↓
Primary Hypothyroidism	↓	↓	↓ ↔	↑
Secondary Hypothyroidism	↓	↓	↓	↓

↑ = elevated, ↓ = decreased, ↔ normal.

after drug intake. Androgenic steroids, genetic factors, hepatic failure, nephrotic syndrome, and protein malnutrition can all lower the thyroglobulin level and falsely lower T_4.[6]

Free T_4 is difficult to measure directly in whole serum because it is in continuous rapid equilibrium with T_4 bound to serum proteins. T_3 resin uptake (T_3RU), an indirect measure of the unbound plasma concentration of T_4, is inversely proportional to the quantity of T_4-binding proteins and is directly proportional to the degree of saturation of the patient's own binding proteins by endogenous thyroid hormone. The total T_4 and the T_3RU are multiplied to achieve the free thyroxine index, which correlates directly with the metabolic status of the patient.

The serum T_3 is measured by radioimmunoassay. The upper limit of the normal level tends to drop with each decade of life, so a level that is normal in a younger patient may indicate hyperthyroidism in an older patient.

Radioactive iodine uptake (RAIU) takes advantage of the great ability of the thyroid gland to concentrate large amounts of inorganic iodide. Oral administration of radioactive iodine (^{131}I) can be elevated in hyperthyroidism. Dietary iodine deficiency, renal failure, or congestive heart failure can increase RAIU. Elevated free T_4 levels, corticosteroids, and dopamine will decrease RAIU. Thyroid scans show functioning thyroid nodules ("hot"), which are rarely malignant. Nonfunctioning ("cold") tissues are more likely to become malignant.

The radioimmunoassay of TSH can detect patients who are hypothyroid, although it is not sensitive enough to differentiate between hyperthyroid and euthyroid states. Starvation, fever, stress, corticosteroids, T_3, and T_4 can depress TSH levels.

The use of TRH to stimulate the release of TSH from the pituitary gland results in an exaggerated response in hypothyroid patients and a subnormal response in hyperthyroid patients. Age greater than 40 in men, a serious illness, fasting glucocorticoid administration, dopamine administration, and some psychiatric disorders also may blunt the response.[5]

Hyperthyroidism usually is associated with the diffuse multinodular goiter of Graves' disease. There are, however, several causes (Table 2). Major manifestations of hyperthyroidism include weight loss, diarrhea, diaphoresis, warm moist skin, skeletal muscle weakness, heat intolerance, tachycardia, cardiac dysrhythmias, mitral valve prolapse, and high-output congestive heart failure. Patients with Graves' disease may exhibit ophthalmopathy and clubbing. "Thyroid storm" is a life-threatening illness brought on by an exacerbation of hyperthyroidism from stresses such as infection, injury, and surgery. Characteristics of thyroid storm include hyperpyrexia, tachycardia, anorexia, extreme anxiety, striking alterations in consciousness, and cardiovascular instability.

Perioperative concerns for the patient with hyperthyroidism include (1) suppression of thyroid hormone synthesis, (2) inhibition of

Table 2. *Causes of Hyperthyroidism*

Toxic multinodular goiter
Hyperfunctioning thyroid adenoma
Toxic adenoma
Iodide-induced thyroiditis
Iatrogenic (exogenous) thyrotoxicosis
Neonatal thyrotoxicosis
TSH-secretory tumor
Nontumorigenic pituitary-induced hyperthyroidism
Choriocarcinoma or hydatidiform mole
Struma ovarii
Hyperfunctioning thyroid carcinoma

release of preformed hormone, (3) inhibition of the peripheral conversion of T_4 to T_3, (4) blocking the end-organ effects of thyroid hormone, and (5) supportive therapy.[3]

Propylthiouracil and methimazole are thiourea derivatives that inhibit the synthesis of the thyroid hormone. Both drugs decrease the rate of synthesis of T_4 by interfering with the incorporation of iodine into tyrosine residues of thyroglobulin. In addition to blocking hormone synthesis, these drugs also inhibit the peripheral deiodination of T_4 and T_3. These compounds must be given orally and require 6 to 8 weeks to deplete hormone concentration and achieve symptomatic improvement. Granulocytopenia and agranulocytosis are rare but potentially serious side effects, whereas skin rash, nausea, and fever are more benign. Oral iodide supplied as a potassium iodide solution (Lugol's) is usually given about 1 hour after antithyroid drugs to prevent accumulation of iodide in the thyroid gland. Iodide antagonizes the ability of both TSH and cyclic AMP to stimulate hormone release, thus decreasing the release of thyroid hormone, which can be seen in as little as 24 hours. Vascularity of the thyroid gland is also reduced, thereby reducing the size of the gland. Allergic reactions manifested by angioedema and laryngeal edema may become life threatening.[17]

β-Adrenergic blockade is effective in attenuating the end-organ effects of thyroid hormone. Although β-adrenergic blockade does not inhibit hormone synthesis, it does prohibit the peripheral conversion of T_4 to T_3. Preoperative preparation for surgery usually requires 1 to 2 weeks. Propranolol is titrated according to heart rate and should not be used in patients with congestive heart failure, bronchospasm, or abnormalities of left ventricular function. Any elective surgery should be postponed until the patient is euthyroid. In an emergency, propranolol titrated in 0.5 mg IV boluses up to 10 mg or esmolol titrated to a heart rate of less than 90 beats per minute may be effective.

Other drugs used perioperatively include reserpine, guanethidine, and ^{131}I. Both reserpine and guanethidine decrease central nervous system stores of catecholamines. Dexamethasone, with its predominant glucocorticoid action, (8–12 mg three times a day), reduces

Table 3. *Causes of Hypothyroidism*

PRIMARY	SECONDARY
Subtotal thyroidectomy	Neoplasms
Hasimoto's thyroiditis	Therapeutic irradiation
I-131 therapy	Pituitary surgery
Autoimmune	Idiopathic hypopituitarism
Therapeutic irradiation to the neck	Sheehan's syndrome
Cystinosis	
Endemic goiter	
Iodine excess	
Drug induced (lithium carbonate, thiourea drugs, sulfonamides)	
Thyroid agenesis	
Maternal iodide or antithyroid drugs	

thyroid hormone secretion in Graves' disease and reduces the peripheral conversion of T_4 to T_3. Also, a reduction in the mild hypercalcemia seen in hyperthyroidism is achieved through the inhibition of bone resorption. The principal indication for ^{131}I therapy is in apathetic hyperthyroidism, which is usually seen in elderly patients (>60 years) who are asymptomatic except for the cardiovascular system. ^{131}I should not be used during pregnancy, as the fetal thyroid gland concentrates the isotope.

Other preoperative concerns include muscle weakness of the proximal muscle group that usually does not involve respiratory paralysis. Severe diarrhea may necessitate fluid and electrolyte replacement. Mild anemia, thrombocytopenia, increased serum alkaline phosphatase, hypercalcemia, muscle wasting, and bone loss may all be involved in hyperthyroidism. Anticholinergic drugs, especially atropine, should be avoided because they interfere with the sweating mechanism and cause tachycardia. A patient with a large thyroid and a potentially difficult airway should be treated with the caution given any other patient with a difficult airway. Because there is an increased incidence of myasthenia gravis in hyperthyroid patients, caution should be used in the administration of nondepolarizing muscle relaxants. Depolarizing muscle relaxants should be avoided.[13]

Hypothyroidism results from inadequate circulating levels of T_4 and T_3. A relatively common disease, hypofunction of the gland has many causes (Tables 1 and 3). Characteristics of hypothyroidism include slow mental functioning, slow movement, dry skin, cold intolerance, depression of the ventilatory responses to hypoxia and hypercarbia, impaired clearance of free water, slow gastric emptying, bradycardia, and depressed myocardial contractility. Amyloidosis associated with cardiac conduction abnormalities, renal dysfunction, and tongue enlargement is not uncommon. Other abnormalities include reduced platelet adhesiveness, gastrointestinal bleeding, anemia, hypothermia, and impaired renal concentrating ability. In "myxedema coma," a rare and severe form of hypothyroidism, stupor, coma, changes in mental activity, hypoventilation, hypothermia, hypotension, decreased cardiac reserve, slowed gastric emptying, and hyponatremia may be present.

The clinical course may be slow and insidious and only become apparent at the time of surgery. With a high mortality rate (>50%), aggressive therapy with T_3 (50–200 μg) bolus for rapid replacement is indicated. T_3 is probably superior to T_4, as it is the more physiologically active form of thyroid hormone and bypasses the conversion from T_4 to T_3, which is usually depressed in severely ill patients. T_4 (400–500 μg) bolus followed by an infusion of 200 μg daily may be substituted if time allows. Hyponatremia associated with a marked decrease in free water clearance by the kidney can be detected, and supportive therapy must be initiated. Glucocorticoid therapy should be considered because of acute primary adrenal insufficiency. Dosages of 100 to 300 mg/day of hydrocortisone have been suggested. Heart rate, blood pressure, and body temperature should return to normal within 1 day. A relatively euthyroid state takes 3 to 5 days to achieve.

Although controversy exists as to whether a patient should be euthyroid before emergency surgery, it appears that those with mild to moderate hypothyroidism can proceed to surgery without an increasing mortality.[9] Thyroid hormone replacement in patients with ischemic heart disease can precipitate angina, dysrhythmias, or even heart failure. It may be prudent to postpone surgery in severely hypothyroid patients until they receive at least some treatment. Thyroid hormone replacement should be continued on the morning of the surgery.

PARATHYROID GLAND

The parathyroid glands function in the regulation of blood calcium concentration through the synthesis and release of parathyroid hormone. Parathyroid hormone increases blood calcium concentrations by increased bone resorption, increased calcium resorption from gut and soft tissue, and through decreasing renal calcium clearance and increasing phosphate clearance in the proximal tubules. Control of parathyroid hormone stems from calcium, phosphate, magnesium, epinephrine, and metabolites of vitamin D.

Primary hyperparathyroidism becomes increasingly common with age and affects women four times more often than men. Primary hyperparathyroidism and malignancy-associated hypercalcemia account for approximately 90% of all cases of hypercalcemia. Usually this disease is the result of enlargement of a single gland, usually an adenoma and rarely a carcinoma. The cause of the hypercalcemia must be differentiated (Table 4). Ionized calcium is the physiologically active fraction. It is influenced by temperature and blood pH through alterations in calcium protein binding to albumin. Acidosis tends to

Table 4. *Differential Diagnosis of Hypercalcemia*

Multiple myeloma
Lymphomas and leukemia
Bone metastases
Hyperparathyroidism
Hyperthyroidism
Hypothyroidism
Addison's disease
Sarcoidosis
Tuberculosis
Vitamin D and vitamin A therapy
Immobilization
Adrenal insufficiency
Milk alkali syndrome
Diuretic therapy
Lithium therapy
Pheochromocytoma
Familial hypercalcemic-hypocalciuric syndrome

increase the ionized calcium, whereas alkalosis tends to decrease it. Patients with hyperparathyroidism have elevated calcium and low serum phosphate levels. A mild hyperchloremic acidosis may be present. Hypercalcemia resulting from primary hyperparathyroidism affects multiple organ systems. In the kidney, nephrolithiasis and renal calcification leading to renal failure usually occur late in the disease process. The associated hypertension may be an end product of renal artery stenosis. Neuromuscular dysfunction with atrophy of muscle fibers and weakness is noted especially in proximal muscle groups. Muscle atrophy also may occur. Radiographs of the hands can reveal skeletal demineralization, which manifests as osteitis fibrosa cystica and simple diffuse osteopenia. Patients may have a history of frequent fractures or report of bone pain. Mental changes such as psychiatric disturbances, memory impairment, obtundation, and depression may appear. Other electrolyte disturbances include hypophosphatemia and hypomagnesemia. Peptic ulcer disease caused by an increased production of gastrin and gastric acid is seen. Anorexia, vomiting, and constipation also may be present. Hypertension occurs in approximately one third of the patients and usually resolves with therapy of the primary disease. End-organ effects of long-standing hypertension include deposition of calcifications in the myocardium, blood vessels, brain, and kidney.[7]

The most useful test for hyperparathyroidism is the radioimmunoassay of parathyroid hormone. In the hyperparathyroid patient the hormone levels are abnormal for a given level of calcium, inorganic phosphorus in serum is usually low, and alkaline phosphatase is elevated if there is considerable skeletal involvement. The parathyroid hormone level is increased with hypercalcemia in hyperparathyroidism and ectopic parathyroid hormone syndrome. All other causes of hypercalcemia have normal levels of parathyroid hormone.

Secondary hyperparathyroidism is usually a consequence of chronic renal disease. It is accompanied by hypocalcemia and hyperphosphatemia. Cardiac symptomatology such as ischemia, myocardial depression, valvular heart disease, atrioventricular block, and supraventricular dysrhythmias occur because of arterial calcification with obstruction.

Therapy for hypercalcemia includes enhancing urinary calcium excretion and reducing calcium entry into extracellular fluid from bone. Volume expansion with isotonic solutions lowers calcium by increasing glomerular filtration rate and glomerular filtration of calcium. Careful monitoring to prevent fluid overloading and thus congestive heart failure is necessary. Hypokalemia and hypomagnesemia must be corrected. Loop diuretics block calcium reabsorption in the thick ascending limb of Henle only if adequate volume expansion has occurred to allow a proper amount of calcium and NaCl to reach the limb of Henle. Therefore, diuretics must only be used after volume expansion. Electrolyte losses, such as magnesium and potassium, must be monitored and corrected as needed. Phosphate should be given to correct hypophosphatemia, as this abnormality decreases calcium uptake into bone, increases calcium absorption from the intestine, and stimulates breakdown of bone. Mithramycin, an inhibitor of RNA synthesis, is effective in greatly reducing the calcium plasma concentration in 24 to 48 hours by decreasing the responsiveness of osteoclasts to parathyroid hormone. The starting dose is 25 μg/kg IV. Side effects may include nausea, thrombocytopenia, and hepatic and renal toxicity. Calcitonin, a peptide derived from the C cells of the thyroid, reduces calcium levels modestly by inhibiting bone resorption and enhancing urinary calcium excretion. Its side effects include urticaria and nausea. Corticosteroids, such as prednisone, reduce the absorption of calcium from the gastrointestinal tract by antagonizing the actions of vitamin D. The onset of this action is slow and unpredictable. Oral phosphate therapy can be used to correct hypercalcemia for the reasons previously stated. Other remedies such as restriction of calcium intake and weight bearing and renal dialysis for patients with severe renal failure have been effective.[1]

Other preoperative considerations for the patient with hypercalcemia caused by hyperparathyroidism include examination of the electrocardiogram for shortened P-R or Q-T intervals. A history of chronic versus acute hypercalcemia is important in determining the extent of end-organ damage to the cardiac, renal, or central nervous system.

In hypoparathyroidism, a decreased production of the parathyroid hormone results in hypocalcemia. The diagnosis must be differentiated (Table 5). Normally, hypocalcemia causes an increase in parathyroid secretion and 1,25(OH)2 D synthesis with an increase in Ca^{2+} mobilization from bone, gastrointestinal absorption, and renal tubule reabsorption. Parathyroid hormone inhibits renal tubular reabsorption of phosphate and bicarbonate. Serum phosphate and bicarbonate levels are elevated in patients with hypoparathyroidism.

Table 5. *Differential Diagnosis of Hypocalcemia*

- Surgical removal parathyroids
- Chronic renal disease
- Malabsorption syndrome
- Pseudohypoparathyroidism
- Hypomagnesemia
- Osteoblastic metastasis
- Acute pancreatitis
- Hypoalbuminemic states

Hypoparathyroidism is divided into three categories: acquired hypoparathyroidism, idiopathic hypoparathyroidism, and pseudohypoparathyroidism. The most common cause of acquired hypoparathyroidism is the surgical removal of the parathyroid glands. Other causes of hypoparathyroidism include chronic renal insufficiency, malabsorption syndrome, pseudohypoparathyroidism, hypomagnesemia, osteoblastic metastasis to bone, pancratitis, ^{131}I therapy, neck trauma, malignancy, and amyloidosis. The clinical manifestations of the disease are related to the hypocalcemia. Neuronal irritability in the form of tetany, muscle spasms in the face and extremities, and seizures may occur. Laryngeal muscle involvement may cause spasm of the cords, fixing them in the midline position and resulting in stridor and cyanosis. Chvostek's and Trousseau's signs are the two classic signs of latent tetany. Hypocalcemia delays ventricular repolarization, increasing the Q-T interval of the electrocardiogram. The prolonged Q-T interval may also indicate heart block. Congestive heart failure, hypotension, and relative resistance to β-adrenergic agonists may occur. Seizures may develop and may be focal, Jacksonian, petit mal, or grand mal. Therapy with standard anticonvulsants may be ineffective and may even aggravate the condition. Other clinical manifestations include depression; muscle stiffness; paresthesias; dry, scaly skin; brittle nails; and coarse, brittle hair. Therapy depends on the type of hypoparathyroidism. In the acute situation, intravenous administration of calcium gluconate, 10 to 20 mL, 10% solution, over several minutes followed by a continuous infusion of 1 to 2 mg/kg/h is indicated. In chronic hypoparathyroidism, vitamin D with or without supplementation is given. The perioperative goal is to achieve normal levels of calcium, magnesium, and phosphate. Symptoms should be under control before anesthesia and surgery. A preoperative electrocardiogram should be evaluated, as an increase in duration of the S-T segment and Q-T interval can be used as a guide throughout the procedure. The Q-T interval varies with heart rate—a heart rate of 50 to 60 has a normal Q-T interval of 0.39 to 0.41 seconds.[4] Phenothiazines should be avoided because they may precipitate dystonic reactions. Respiratory alkalosis also should be avoided, as this may exacerbate neuronal irritability.[8]

ADRENAL GLAND

The adrenal cortex is the site of synthesis and secretion of three major classes of hormones: mineralocorticoids, glucocorticoids, and androgens. The excess or deficiencies of any of these hormones causes a clinical syndrome requiring special consideration. Steroid supplementation, aging, and certain anesthetics play an important role in preparation for an anesthetic.

The glucocorticoid cortisol is the essential regulator of carbohydrate, protein, lipid, and nucleic acid metabolism. Cortisol is bound to transcortin, although the unbound portion enters cells and is active. Daily cortisol production reaches 20 mg and decreases about 30% in the elderly. Cortisol is produced in the zona reticularis under the control of adrenocorticotropic hormone (ACTH), which is synthesized and released from the anterior pituitary gland. ACTH is controlled by the hypothalamus through the release of corticotrophin-releasing factor (CRF). Cortisol and the other glucocorticoids exert a negative feedback at the pituitary and hypothalamic levels to inhibit the secretion of ACTH and CRF. Glucocorticoids enhance gluconeogenesis, elevate blood glucose, and promote hepatic glycogen synthesis.

Aldosterone, the major mineralocorticoid produced by the zona glomerulosa, causes reabsorption of sodium and secretion of potassium and hydrogen ions, thus contributing to electrolyte and volume homeostasis. The main regulator of aldosterone secretion is the renin-angiotensin system. Renin separates the precursors angiotensinogen into angiotensin I which is further converted by converting enzyme in the lung to angiotensin II.

Glucocorticoid excess (Cushing's syndrome) can be caused by endogenous oversecretion of glucocorticoids caused by bilateral adrenal hyperplasia secondary to ACTH produced by an anterior pituitary microadenoma or nonendocrine tumor such as lung, kidney, or pancreas. Another cause for the excess is exogenous administration of high doses of glucocorticoids for other disease processes. The symptoms are typical and include truncal obesity, moon-facies, hypertension, hyperglycemia, increased intravascular fluid volume, hypokalemia, easy fatigability, thin skin with easy bruisability, abdominal striae, osteoporosis with some pathologic fractures, and muscle weakness. The diagnosis is made by observing elevations in plasma and urinary cortisol, urinary 17-hydroxycorticosteroids, and plasma ACTH levels. Failure to suppress a low-dose dexamethasone suppression test normally confirms the diagnosis. Metyrapone suppression differentiates adrenal hyperplasia from adrenal tumors. In addition, magnetic resonance (MR) imaging has been used to evaluate the pituitary gland.[2]

Preoperative concerns include correction of fluid and electrolyte status, control of hypertension, and regulation of diabetes. Restricting sodium intake (<80 mEq/day) along with administration of spironolactone (200–400 mg/day), an aldosterone antagonist, stops potassium loss and helps mobilize excess fluid. Amiloride (10–40 mg/day) may be used for sexually active males intolerant of spironolactone because amiloride blocks sodium channels at the distal renal tubule thus increasing sodium and chloride excretion and decreasing potassium excretion. Calcium channel blockers have been effective because calcium is the final common pathway for aldosterone production. Angiotensin-converting enzyme inhibitors return blood pressure to normal, decrease aldosterone secretion rates, and improve potassium balance.[18] A hypokalemic alkalosis may be produced from ectopic ACTH production from sites other than the pituitary gland.[14] Attention to positioning and sterility during surgery must be meticulous to prevent fractures and infections in these highly susceptible patients. Peak pulmonary status must be achieved in those patients undergoing bilateral adrenalectomy, as pneumothorax occurs in 5% to 10% of these patients. Intraoperative monitoring of central venous pressures should be considered for extensive procedures as should pulmonary artery monitoring in those patients whose cardiac status is not optimal. Glucocorticoid administration should start at the beginning of adrenal resection. Hydrocortisone phosphate, 100 mg IV every 12 hours, is the recommended dose to be continued for 3 to 6 days. Therapy is individualized based on the absence or presence of any remaining adrenal tissue.

Mineralocorticoid excess (primary hyperaldosteronism or Conn's syndrome) leads to sodium retention, potassium depletion, hypertension, and hypokalemic alkalosis. Intravascular fluid volume, electrolyte concentration, and renal function should be restored preoperatively. Spironolactone should be given 1 to 2 weeks in advance because of its slow onset of the action. Pulmonary artery monitoring should be considered because of the increased incidence of ischemia in these patients.

Adrenocortical insufficiency follows abrupt withdrawal of steroids, destruction of the adrenal gland by cancer, tuberculosis, hemorrhage, an autoimmune mechanism (e.g., Hashimoto's thyroiditis), or administration of cytotoxic drugs. Primary adrenal insufficiency (Addison's disease), caused by destruction of the gland, usually develops slowly and produces marked pigmentation and cardiac dysfunction secondary to chronic hypotension. Secondary adrenal insufficiency results from decreased ACTH secretion from the anterior pituitary. Pituitary failure may be caused from tumor, infection, surgical ablation, or radiation therapy, and symptoms includes weight loss, anorexia, abdominal pain, nausea, vomiting, diarrhea, and constipation. Hyperkalemia may cause life-threatening dysrhythmias. It is diagnosed by evaluating the response to synthetic ACTH administration. Little or no response by the adrenal glands is considered diagnostic of hykalemia. The metyrapone suppression test may suggest an upcoming adrenal crisis in patients with adrenal insufficiency. Preoperative therapy includes glucocorticoid therapy (300 mg hydrocortisone/70 kg/day) and the mineralocorticoid fludrocortisone (0.05–0.1 mg/day) along with correction of hypovolemia, hyperkalemia, and hyponatremia. Acute adrenal insufficiency (crisis) can be precipitated by sepsis, trauma, or surgical stress. Immediate therapy with steroid replacement along with fluid and electrolyte resuscitation should be instituted.

Hypoaldosteronism may be caused by unilateral adrenalectomy, prolonged administration of heparin, and long-standing diabetes with renal failure or it may be congenital. Symptoms includes hypotension, hyperkalemic acidosis, hyperkalemia, hyponatremia, and myocardial defects. Fludrocortisone therapy (0.05–0.1 mg/day) with electrolyte correction is needed.

The decision whether to supply steroid supplementation for patents taking steroids for other reasons has been widely debated. The most sensitive test of adrenal reserve is the ACTH stimulation test in which a normal response of plasma cortisol levels of greater than 18 μg/dL 60 minutes after administration of synthetic ACTH indicates return of normal pituitary-adrenal axis function. The potential risks of perioperative supplementation include aggravation of hypertension, hyperglycemia, fluid retention, stress ulcers, psychiatric disturbances, abnormal wound healing, and an increased rate of infection. The precise amount of glucocorticoid required has not been established and many regimens exist. I suggest supplementing any patient who has had steroids at any time during the year prior to surgery, even if in topical application. Hydrocortisone phosphate, 100–200 mg/70 kg/day IV, is given just prior to surgery.[10]

Pheochromocytoma is a tumor of chromaffin cells located in the adrenal medulla. These cells are of neural crest origin and are capable of secreting neurotransmitters such as epinephrine, norepinephrine, dopamine, serotonin, histamine, and melatonin. Physiologically active peptides such as endorphins, enkephalins, renin and converting enzyme, vasopressin, and thyroid-releasing hormone are also produced. Eighty-five percent of these tumors are solitary. About 5% are bilateral and 10% are extraadrenal, that is, occurring anywhere (base of the brain, testicle, and the interatrial cardiac septum). The size, weight, or histologic appearance of the tumors are variable and bear little relationship to the symptoms. Malignant spread, which occurs in fewer than 15%, proceeds by venous and lymphatic channels and favors the liver. Pheochromocytomas may arise in association with multiple endocrine neoplasia Type IIA or IIB, pancreatic islet cell tumors, and neuroectodermal disorders such as neurofibromatosis and von Hippel-Lindau disease. Paroxysmal hypertension places patients at risk for cerebrovascular hemorrhage, heart failure, dysrhythmias, or myocardial infarction. Orthostatic hypotension from hypovolemia is common. Clinically, the triad of paroxysmal sweating, hypertension, and headache is a strong indication for measurement of urinary excretion of norepinephrine, epinephrine, dopamine, total metanephrines, and vanillylmandelic acid (VMA) along with plasma concentration of norepinephrine and epinephrine. Urine determinations are made on samples collected during a 24-hour period, and urinary VMS and unconjugated norepinephrine and epinephrine are expressed as hourly rates or per milligram of creatinine. VMA levels (>9.0 mg/24 h), norepinephrine (>170 μg/24 h), and epinephrine (>35 μg/24 h) affords 95% clinical specificity and sensitivity.[15] Localization of the tumor with computed tomography and magnetic resonance imaging has proved effective. Scintigraphic localization using ^{131}I-metaiodobenzylguanidine (MBIG), echocardiography, and or biochemical testing from sequential sampling for plasma catecholamines through the inferior and superior vena cava systems should be considered in locating the tumor. The possibility of multiple tumors should be considered and other sites also examined at surgery, if feasible.

Perioperative considerations for patients undergoing excision of a pheochromocytoma include controlling hypertension, hyperglycemia, and fluid status without compromising cardiac status. α-Adrenergic blockade should be established before surgery. Phenoxybenzamine, a long-acting noncompetitive α-1 and α-2 blocker at doses of 10 mg every 8 hours is given. The dosage should be tapered for tachycardia. Treatment may be necessary for 10 to 14 days or until symptoms are alleviated. Optimal preoperative conditions have not been firmly established but current recommendations include (1) blood pressures no higher than 160/90 mm Hg for at least 24 hours before surgery, (2) little orthostatic hypotension with readings above 80/45 mm Hg, (3) electrocardiographic readings free of ST-T changes for at least 1 week, and (4) no more than one premature ventricular contraction every 5 minutes.[11] Prazosin, an α-1 blocker, also has been used to establish alpha blockade. Labetalol, an alpha and beta blocker, has been successfully used preoperatively to control blood pressure. After the alpha blockade is established, beta blockade can be established for patients who have persistent tachycardia or dysrhythmias to avoid the potential for unopposed alpha-mediated vasoconstriction and worsening of the hypertension. An acute hypertensive crisis can be treated with intravenous infusions of nitroprusside or phentolamine. Phentolamine, a short-acting alpha-agonist, has a rapid onset (within 2 minutes) and lasts only 10 to 15 minutes. Declines in blood pressure cause baroreceptor-mediated increases in sympathetic nervous system activity, which causes a tachycardia and cardiac dysrhythmias. Esmolol, a short-acting beta blocker can be used to control these symptoms. Central venous and peripheral artery pressure monitoring is used to guide preoperative fluid and cardiac status.

DIABETES MELLITUS

Diabetes mellitus is the most common endocrine disease that concerns the anesthesiologist. End-organ involvement results in cataracts, retinopathy, peripheral neuropathy, nephropathy, and angiopathy. Acute complications for the diabetic patient include hypoglycemia, diabetic ketoacidosis, and hyperglcemic, hyperosmolar coma.

Preoperative evaluation of diabetic patients should include a thorough history to elicit their therapeutic, dietary, and possible exercise regimens. Laboratory testing of blood and urine will detail the ade-

Table 6. *Sliding Scale Regimen*

BLOOD GLUCOSE (mg/dL)	REGULAR INSULIN SQ (units)
200–250	2–3
250–300	3–4
300–350	4–5
350–400	5–8*
>400	8–10*

* Check glucose levels every hour.

quacy of glucose control. Previous surgical and anesthetic exposures and or complications should be discussed. The presence of any end-organ complications such as cardiac disease, renal insufficiency, peripheral neuropathy, or peripheral vascular disease should be detailed. Special care should be taken to practice aseptic technique because of the diabetic patient's increased susceptibility to infection. Serial electrocardiograms and or information from the patient's cardiologist may help to determine the cardiac reserve. Patients with significant autonomic neuropathy may be at increased risk of aspiration from gastroparesis and may have an increased incidence of cardiorespiratory arrest on impaired respiratory responses to hypoxia. Susceptibility to the action of depressant drugs is increased. Preoperative fluid resuscitation with Ringer's lactate may worsen existing hyperglycemia by increasing the lactate load and metabolic acidosis.

Several basic preoperative objectives include (1) achieving good control of blood glucose levels with correction of any acid-base, fluid, or electrolyte abnormalities; (2) providing an adequate amount of carbohydrate to inhibit catabolic proteolysis, lipolysis, and ketosis; (3) providing insulin adequate to prevent hyperglycemia, glycosuria, and ketoacidosis while avoiding hypoglycemia; and (4) monitoring the end organs that are involved such as the heart, kidney, and brain. Short-acting oral hypoglycemic drugs are omitted on the day of surgery, and long-acting drugs may be discontinued 2 days before surgery or continuous IV infusions of glucose may be started. Usually, one half the dose of intermediate-acting insulin is given subcutaneously *after* an IV infusion of 5% dextrose. Blood glucose levels must be obtained frequently. Regular insulin should be administered intravenously as determined blood glucose levels and by using a "sliding scale" (Table 6). Some anesthesiologists prefer not to give any insulin on the morning of surgery and continue glucose containing solutions, especially in high-risk diabetic patients who require a continuous intravenous infusion of regular insulin of 1 to 2 units per hour.

Many diabetic patients require emergency surgery for trauma or infection and may have diabetic ketoacidosis. If at all possible insulin therapy and fluid resuscitation are initiated and electrolytes corrected prior to commencing surgery. Potassium replacement may be needed, as total body potassium is depleted even more during hydration. Phosphorus deficiency in ketoacidosis may result in significant muscular weakness and organ dysfunction. The degree of acidosis is determined by measurement of the anion gap $[Na^+ - (Cl^- + HCO3)]$. Acidosis with an increased anion gap (>16 mEq/L) may be caused by ketones in ketoacidosis, lactic acid in lactic acidosis, or increased organic acids from renal insufficiency. Bicarbonate therapy in these situations is controversial. Myocardial function and respiration may be depressed. The development of cerebral spinal fluid and central nervous system acidosis from rapid conversion of bicarbonate to carbon dioxide with diffusion of the acid across the blood-brain barrier, with altered CNS oxygenation and the resultant decreased cerebral blood flow, and with the development of unfavorable osmotic gradients may result in alterations in central nervous system function and structure.[16]

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Preanesthetic Assessment of the Patient with Gastrointestinal Disease

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The gastrointestinal system, including the liver and pancreas, serves as a vital interface between the human body and its external environment. It is responsible for the intake and processing of life-sustaining nutrients, electrolytes, and water. Although the emphasis in preanesthetic assessment of the surgical patient is usually placed on cardiovascular and respiratory function, pathologic conditions of the gastrointestinal tract may result in life-threatening acid-base, fluid, and electrolyte abnormalities. The anesthesiologist evaluating such a patient must be aware of the far-reaching implications of gastrointestinal disease in general as well as specific problems associated with various disease entities.

GENERAL CONSIDERATIONS

The Full Stomach and Aspiration

Aspiration of gastric contents into the lungs is one of the most feared anesthetic complications and remains a major cause of morbidity and mortality in anesthesia.[18] During the maintenance of routine anesthesia, the incidence of gastric contents reaching the perilaryngeal area is approximately 8% and regurgitated material reaches the airway in 1% of the population.[3] Solid matter may obstruct the airway, whereas nonacid liquid may produce hypoxemia through bronchospasm and atelectasis. Aspiration of even small amounts of acidic liquid may be associated with Mendelson syndrome, a severe chemical pneumonitis, resulting in bronchospasm, hypoxemia, and atelectasis, with possible progression to adult respiratory distress syndrome (ARDS) and pulmonary edema. The incidence of Mendelson

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syndrome has been estimated at 0.02% (1 in 4521 anesthetics administered).[16]

A gastric volume greater than 25 mL and a gastric pH less than 2.5 are associated with an increased risk of aspiration pneumonitis.[22] The patient with gastrointestinal disease may meet both criteria. Obesity, a history of gastritis or ulcer, and emergency abdominal surgery are among the factors indicating a nonempty stomach and an increased risk of aspiration.[20] Incompetence of the lower esophageal sphincter (LES), delayed gastric emptying, and increased intragastric pressure all predispose the patient to aspiration.

Several approaches minimize the risk of aspiration. Reduction of gastric volume and pH are important, as even aspirates with a pH greater than 2.5 may induce an acid aspiration syndrome.[6] The efficacy of maintaining a fasting period has been challenged in elective surgical patients,[15, 16] and it is certainly inadequate protection in the patient with gastrointestinal disease in whom decreased gastric motility, increased gastric pressure, increased volume with decreased pH, or an incompetent LES may be complicating factors. Regional anesthesia is generally considered safer with regard to the aspiration risk. Patients with gastrointestinal disease, however, often present for procedures requiring general anesthesia. Additionally, the anesthesiologist must always be prepared to convert a failed or otherwise complicated regional anesthetic to a general anesthetic. Thus, other means of aspiration prophylaxis are recommended.

The stomach may be mechanically emptied using nasogastric suctioning or by induction of vomiting with an emetic such as apomorphine. Neither approach, however, is completely effective in emptying the stomach. Induced vomiting is unpleasant, and there is a small risk of esophageal laceration. Apomorphine may produce cardiovascular collapse in ill patients and those with toxic conditions.[14] If a nasogastric tube is in place before induction, some anesthesiologists believe it should be removed because it may serve as a wick and allow passive reflux and act as a mechanical hinderance to endotracheal intubation.[21] Other studies have demonstrated the efficacy of cricoid pressure in preventing aspiration despite the presence of a nasogastric tube.[25]

Presurgical administration of oral antacids effectively raises gastric pH. Because particulate antacids may themselves induce a pulmonary inflammatory response when aspirated,[10] the trend is toward using nonparticulate antacids such as sodium citrate (0.3 mM) or similar commercial preparations.

H_2 receptor antagonists such as cimetidine and ranitidine reduce acid secretion, increase gastric pH, and may decrease gastric volume. Administration may be orally several hours preoperative (cimetidine 300 mg, ranitidine 150 mg) or intravenously 45 to 60 minutes preoperative (cimetidine 300 mg, ranitidine 50 mg). An additional dose the night before surgery may improve effectiveness.[2, 29] Anticholinergics, especially glycopyrrolate, reduce gastric acidity but decrease LES tone.

Metoclopramide, 10 mg IV 15 to 30 minutes before induction, increases LES tone, decreases gastric fluid volume, and acts as an antiemetic.[11] Omperazole, a new H^+K^+ATPase inhibitor, decreases gastric acidity and may be useful in the future.

Although preoperative prophylaxis may decrease the likelihood of aspiration and its sequelae, there is no substitute for a properly planned endotracheal intubation to secure the airway. Rapid sequence intubation with cricoid pressure (Selick maneuver) is the preferred technique for airway protection in the full-stomach situation. The anesthesiologist must be relatively certain that the patient's airway will not prove difficult to intubate. If there is uncertainty about the anesthesiologist's ability to secure the airway, an awake intubation with good topical anesthesia may be more appropriate. Superior laryngeal nerve or transtracheal blocks may improve patient compliance but also compromise laryngeal reflexes.

Metabolic Abnormalities in Gastrointestinal Disease

Gastrointestinal disease leads to fluid, electrolyte, nutritional, and acid-base disturbances by several mechanisms. Patients with a pathologic gastrointestinal condition may be unwilling or unable to maintain a normal nutrient and fluid intake. While awaiting surgery, prolonged fasting periods without adequate parenteral replacement may result in dehydration. Malnutrition may develop rapidly when gastrointestinal abnormalities such as malabsorption, malignancy, obstruction, dysphagia, or prior gastrointestinal tract surgery exist. Wound healing and immune function are compromised in the malnourished surgical patient.[24]

Vomiting is commonly associated with many gastrointestinal disease states and results in diminished extracellular volume from the loss of fluids and electrolytes. Protracted vomiting results in more serious metabolic consequences depending on the quantity and composition of the vomitus. If the loss is primarily gastric juice, then large amounts of hydrogen, chloride, sodium, and potassium ions are lost. The loss of acid causes a metabolic alkalosis with accumulation of bicarbonate ions in the blood. The potassium deficit sustains the extracellular alkalosis, as potassium ions move out of cells while hydrogen ions move in. Because of the elevated plasma HCO_3^- concentration, the HCO_3^- load filtered by the kidney is increased as is delivery of $NaHCO_3$ to the distal tubule. The aldosterone response initiated by hypovolemia increases resorption of $NaHCO_3$ in exchange for K^+ and H^+. This paradoxic aciduria (in the face of metabolic alkalosis) and potassium loss further aggravate alkalosis and hypokalemia. Similar consequences result from prolonged nasogastric suctioning.

If significant amounts of pancreatic juice and bile are lost, the shift in acid-base balance may be toward metabolic acidosis, as these solutions have alkaline pH ranges. In this instance, H^+ moves into cells in exchange for K^+, thus increasing plasma potassium levels.

Because of increased urinary potassium loss, however, total body hypokalemia follows.

Diarrhea may be a consequence of impaired absorption of water and electrolytes, an increase in osmotically active substances in the bowel lumen, or abnormal intestinal motility. Diarrheal stools are usually isotonic or somewhat hypotonic, and volume loss may be great (1–4 L/day). The loss of potassium and bicarbonate causes a hypokalemic metabolic acidosis.

Gastrointestinal fistulas are abnormal communications between the stomach or intestines and either the skin, another portion of intestine, or a viscus such as the bladder or gallbladder. They may arise secondary to surgical procedures, a tumor, bowel inflammation, or ischemic necrosis of the bowel. They frequently are associated with great volume losses and electrolyte imbalances similar to those seen in diarrhea, malnutrition, and infection.

In small bowel obstruction, fluid and gas rapidly accumulate in the bowel lumen. The fluids result from decreased absorptive capacity and increased intestinal secretions. As dilatation increases, fluid accumulates in the bowel wall and peritoneal cavity. The raised intraluminal pressure impairs blood supply and increases the potential for necrosis and perforation. In this emergency, additional fluid loss leads to hypovolemic shock, which may be complicated by bacterial toxemia or septicemia.

Obstruction of the large bowel proceeds more slowly than in small bowel obstruction. The consequences of the obstruction depend on the competence of the ileocecal valve. If the valve is competent, a closed loop obstruction dilates the large bowel and creates the potential for ischemic necrosis and perforation. If the valve is not competent, the bowel contents reflux into the small intestine resulting in dilatation and feculent vomiting.

Clearly, most patients with gastrointestinal disease require a thorough preoperative evaluation and correction, as necessary, of acid-base, fluid, electrolyte, and nutritional status.

Assessment of volume deficit should begin with physical examination. Clinical manifestions of a hypovolemic state may include diminished skin turgor, dry mucus membranes, reduced blood pressure, central venous pressure and urine output, postural hypotension, peripheral cyanosis from sluggish circulation, vasoconstriction, and desaturation of venous blood. Gastrointestinal volume loss, e.g., by vomiting or diarrhea, is usually isotonic in nature. If water is lost in excess of sodium, plasma sodium concentration is increased, as occurs when gastrointestinal losses are compounded by imperceptible loss such as sweating in the febrile patient unable to drink. Conversely, if hypotonic solutions are administered to such a patient, hyponatremia may result. In prerenal azotemia, the blood urea nitrogen increases disproportionately to the serum creatinine (normal ratio, 10–15:1). When volume depletion is severe enough to reduce the glomerular filtration rate, the creatinine increases and the ratio is maintained. Reduction

Table 1. *Gastrointestinal Causes of Electrolyte Disorders*

- Hyponatremia
  - Vomiting/GI suction
  - Diarrhea
  - Fistulae
  - Ileus/obstruction
  - Peritonitis
  - Pancreatitis
  - Drainage of ascites
  - Gastric aspiration
- Hypomagnesemia
  - Vomiting
  - Diarrhea
  - Fistulae
  - Pancreatitis
  - Alcoholic cirrhosis
  - Malabsorption: short bowel syndrome
    - bypass surgery for obesity
  - Gastric aspiration
  - Protein-calorie malnutrition
- Hypokalemia
  - Deficient dietary intake
  - Vomiting/GI suction
  - Fistulae
  - Malabsorption
  - Ureterocolostomy
  - Villous papilloma
  - Gastric aspiration
  - Laxative abuse
  - Ion exchange resins
- Hypocalcemia
  - Extensive bowel resection
  - Antacid therapy
  - Acute pancreatitis
  - Generalized peritonitis

Data from McCloy RF: Metabolic problems associated with gastrointestinal and pancreatic disease. Br J Anaesth 56:83, 1984.

in plasma volume because of volume depletion tends to elevate the hematocrit level and plasma albumin concentration. Invasive monitoring of central venous or pulmonary artery pressure further assists in the assessment of volume status and resuscitation.

Serum electrolyte concentrations should be maintained, as electrolyte imbalances are common in gastrointestinal disease (Table 1). Hypokalemia may produce disturbances of myocardial conduction, rhythmicity and contractility, skeletal muscle weakness, and paralytic ileus. Hypoalbuminemia reflects protein malnutrition. Hypomagnesemia may be seen with protracted vomiting or diarrhea. Manifestations of hypomagnesemia include central nervous system irritability, skeletal muscle spasm, and cardiac dysfunction. A review of the electrocardiogram may reveal changes associated with electrolyte abnormalities.

Arterial blood gas analyses are useful if acid-base derangements are suspected. Metabolic acidosis can impair myocardial function, increase peripheral vasoconstriction, and cause cardiovascular collapse. Metabolic alkalosis can affect myocardial function and also shifts the oxyhemoglobin dissociation curve to the left, impairing tissue oxygenation. Acid-base abnormalities also influence the action and disposition of many anesthetic agents, including narcotics, local anesthetics, thiopental, and neuromuscular blockers. Morphine, meperidine, and fentanyl are weak bases that become less ionized as the pH rises. Alkalinization of the blood thus facilitates narcotic passage into the brain.[19] Respiratory acidosis, which decreases the nonionized fraction of these drugs, nevertheless results in higher brain levels of morphine.[7] This indicates that pH-induced increases in cerebral blood flow and decreases in protein binding are more important than the ionization of the drug. Local anesthetics are also weak bases. When injected into an acidotic environment they give a poor quality of anesthesia because of an increased ionized fraction of drug. The pKa of thiopental is near blood pH (pKa 7.6). Thus acidosis favors the nonionized fraction of the drug and deepens barbituate anesthesia; alkalosis has the opposite effect. Respiratory acidosis enhances d-tubocurarine, pancuronium[17] and vecuronium[19] induced neuromuscular blockade.

The speed with which fluid and electrolyte disorders should be corrected depends on the severity of the disorder and the urgency of the surgery. The immediate goal of therapy is to bring the patient out of danger. Gradual correction of volume deficits minimizes the risk of volume overload and pulmonary edema. In a surgical emergency such as a strangulated small bowel obstruction, however, resuscitation must be swift and aggressive to render the patient fit for the operation.

In hypokalemic patients, the necessity for postponement of elective surgery and aggressive potassium replacement has been questioned. Hypokalemic patients are believed to be at increased risk for dysryhthmias during surgery, and hazardous concentrations have been suggested but not confirmed.[27] Chronic hypokalemia (2.6 mEq/L^{-1}–3.5 mEq/L^{-1}) in asymptomatic elective surgical patients was not found to increase the incidence of intraoperative dysrhythmias.[28] Again, the decision to proceed and the need for supplementation depends on clinical signs and symptoms, the acuteness or chronicity of the potassium changes, and the urgency of the surgical procedure.

SPECIFIC GASTROINTESTINAL DISORDERS

Esophageal Disorders

Esophageal disease predisposes to aspiration, and precautions, therefore, must be taken.[20] Carcinoma of the esophagus leads to dysphagia, malnutrition, and weight loss. The extent of tumor invasion should be known as extension into the trachea and bronchial tree may impair intubation and place the patient at risk for obstruction and bleeding.

Bowel Disorders

Fluid and electrolyte changes in small bowel obstruction and the risk of aspiration have been discussed. Elevation of the diaphragm by a distended bowel may lead to respiratory embarrassment. Sudden decompression of the bowel can result in rapid dilatation of splanchnic venous beds causing hypotension and shock. If bowel obstruction has compressed the inferior vena cava, sudden decompression may lead to a hyperdynamic state and even cardiac failure. Therefore, decompression is best performed gradually, allowing time for hemodynamic compensation. Nitrous oxide diffuses into gas-containing spaces faster than nitrogen leaves because of the former's greater solubility. Prolonged administration of nitrous oxide should be avoided in intestinal obstruction, as distention and intraluminal pressure are increased.

Cholinesterase inhibitors such as neostigmine increase bowel activity. There is anecdotal evidence that in patients with bowel anastamosis, administration of neostigmine may lead to anastamotic disruption, an effect diminished by atropine and abolished by halothane. Neostigmine should be used cautiously in such circumstances.[1]

Ulcerative colitis may result in fluid and electrolyte imbalance, vitamin B_{12} and folate deficiency, and extracolonic manifestations such as arthritis and liver disease. Acute complications include toxic megacolon, perforation, obstruction, and hemorrhage. Patients with Crohn's disease are often chronically ill, malnourished, and dehydrated. Extracolonic manifestations and toxic megacolon are less common than in ulcerative colitis. Fluid, electrolyte, and nutrient loss can occur secondary to malabsorption, fistulae, or obstruction. Patients with inflammatory bowel disease are often maintained on immunosuppressive agents and steroids and require perioperative stress dosing.

Pancreatic Disorders

Pancreatic insufficiency may result in protein and fat malabsorption, hyperglycemia, hypocalcemia, fluid deficit, and hypokalemia. Pleural effusions, adult respiratory distress syndrome, and renal failure may occur in acute pancreatitis. Because pancreatitis is often associated with alcohol abuse, the anesthesiologist must be alert for evidence of withdrawal, liver disease, and malnutrition.

Carcinoid Syndrome

Carcinoid tumors are rare tumors arising from enterochromaffin tissue that is usually found in the gastrointestinal tract. Carcinoid syndrome occurs when these tumors secrete a variety of vasoactive substances including serotonin, prostaglandins, histamine, kallikreins, and bradykinin. Only about 5% of patients with carcinoid tumors develop carcinoid syndrome,[30] which occurs when the release of vasoactive substances overwhelms the liver's ability to inactivate them. Most of these patients have liver metastases that allow these hormones direct access to the systemic circulation. The syndrome is marked by episodes of flushing and cyanosis, bronchospasm, hypotension or hypertension, diarrhea, and right-sided valvular disease of the heart.

Preoperative management involves assessment of symptoms and possibly treatment with drugs that block the effects of the released substances (Table 2). Corticosteroids may prevent bronchospasm and hypotension. H_1 and H_2 blockers oppose histamine effect. Cyproheptadine has both antiserotonin and antihistamine activity. Aprotinin is a kallikrein inhibitor that blocks bradykinin synthesis. Ketanserin, a serotonin receptor antagonist, may be used to prevent or treat bron-

Table 2. *Hormones Released by Carcinoid Tumors and Their Management*

		TREATMENT		
	PHYSIOLOGIC EFFECTS	*Inhibiting Synthesis*	*Hormone Depletion*	*Receptor Blockers*
Serotonin	Vasoconstriction Vasodilatation Increased motility Tryptophan depletion	Parachlorophylamine	Fenluranine	Methysergide Cyproheptadine Ketanserin
Kinins	Vasodilation Histamine release Bronchoconstriction	Steroids Aprotinin		
Histamine	Vasodilatation H_1 = extravascular smooth muscle contraction H_2 = extravascular smooth muscle relaxation			H_2 antagonists Phenothiazines

From Buckley FP: Anesthesia and Obesity and Gastrointestinal Disorders. *In* Barash PG, Cullen BF, Stoelting RK (eds): Clinical Anesthesia. Philadelphia, J.B. Lippincott, 1989; with permission.

chospasm and hypertension. Hypertension also may be treated with methotrimeprazine, a phenothiazine derivative, or the conventional vasodilators sodium nitroprusside and nitroglycerin. Because hypotension may stimulate the release of vasoactive substances, deep planes of anesthesia and regional techniques causing sympathetic blockade should be avoided. Adequate volume should be given to avoid hypotension. Catecholamines and catecholamine releasing substances activate tumor kallikrein and should be avoided. If hypotension does not respond to volume, angiotensin and methoxamine are recommended. Drugs causing histamine release (e.g., morphine, d-tubocurarine) should not be used. In addition to histamine release, succinylcholine may raise intra-abdominal pressure, releasing hormones from the tumor.

Acquired Immunodeficiency Syndrome (AIDS)

Diarrhea, vomiting and loss of appetite commonly occur in the AIDS patient. Oral candidiasis makes eating and swallowing difficult and painful. Cytomegaloviral colitis and proctitis cause debilitating diarrhea. Other intestinal infections are caused by *Cryptosporidium* and *Mycobacterium avium* and *M. intracellulare.*

Thus, the majority of AIDS patients are dehydrated, in negative nitrogen balance, and have electrolyte abnormalities. Preanesthetic correction is essential whenever time permits.

Diseases of the Liver and Biliary Tract

To assess the patient with hepatic dysfunction properly, the anesthesiologist must be aware of the multiple physiologic functions of the liver. The liver maintains glucose homeostasis through glycogenolysis and gluconeogenesis. It is responsible for fat metabolism and the formation of bile salts essential for lipid and fatty acid absorption from the bowel. After hemoglobin is metabolized in the reticuloendothelial system to bilirubin, it is conjugated in the liver and either excreted in the bile or absorbed in the blood stream. The liver synthesizes albumin and most of the clotting factors. Of particular importance in the perioperative period is the liver's role in detoxification and removal of exogenous pharmacologic agents.

The patient with liver disease may have a history of chronic and recent alcohol abuse, gastrointestinal bleeding, easy bruising, anorexia, weight loss, fatigue, nausea, vomiting, diarrhea, and fever. Physical examination may reveal tender hepatomegaly, jaundice, and in the cirrhotic, numerous stigmata including palmar erythema, spider angiomata, Dupuytren's contractures, hypertophied parotid and lacrimal glands, and in men, gynecomastia, and testicular atrophy. The most serious complication of cirrhosis is portal hypertension, which may lead to ascites, splenomegaly with pancytopenia, and bleeding from esophageal varices. Ascites causes abdominal distention and raises the diaphragm, decreasing the functional residual capacity, increasing atelectasis and shunting, and leading to ventilatory insufficiency. Cirrhotic patients often present with a hyperdynamic circulation and a low-normal arterial pressure. This is believed to result from splanchnic circulatory shunts, mesenteric vasodilatation, and possibly, vasodilator substances.[23]

Hepatic encephalopathy may result in personality changes, hyperreflexia, asterixis and altered levels of consciousness, cerebral edema, increased intracranial pressure, and death. The cause is unknown, but it is associated with high plasma levels of abnormal nitrogenous substances such as ammonia.

Blood tests must include hemoglobin and hematocrit assessment, as a complex anemia is frequently seen. Contributing factors to blood content abnormalities may include gastrointestinal bleeding, increased plasma volume, decreased red cell formation, hemolysis, sequestration of blood cells in the spleen and vitamin deficiencies. Leukopenia and thrombocytopenia may reflect hypersplenism and alcohol-induced bone-marrow depression. Progressive renal failure that increases blood urea nitrogen and creatinine levels is an ominous complication of advanced hepatic disease. Hyponatremia and hypokalemia from urinary losses in patients with ascites are commonly caused by secondary hyperaldosteronism and increased antidiuretic hormone. Hypoglycemia may reflect glycogen depletion from malnourishment or impaired gluconeogenesis. A diabetic type of glucose intolerance may reflect insulin resistance in some cirrhotic patients. Hypoalbuminemia results from synthetic dysfunction. Hypomagnesemia is common because of dietary deficiency and urinary losses. Arterial blood gas analysis may reveal arterial hypoxemia, respiratory alkalosis from hyperventilation often present in cirrhotic patients, or metabolic acidosis from impaired lactic acid clearance and advanced renal failure. Prolonged prothrombin time and partial thromboplastin time indicate clotting factor deficiencies. Transaminase (SGOT and SGPT) and lactate dehydrogenase elevations are indicators of hepatocellular dysfunction, whereas alkaline phosphatase and direct bilirubin are a measure of biliary excretion. In late stages of cirrhosis, fibrosis of the liver parenchyma is so widespread that liver enzyme levels decrease to a "normal" range. The most widely used classification of hepatic dysfunction is that of Child and Turcotte (Table 3). Several studies have found Child's class to be predictive of operative

Table 3. *Child's Classification*

	CLASS A	CLASS B	CLASS C
Bilirubin (mg/dL)	<2.0	2.0–3.0	>3.0
Albumin (g/dL)	>3.5	3.0–3.5	<3.0
Ascites	None	Easily controlled	Poorly controlled
Encephalopathy	None	Mild	Advanced
Nutritional status	Excellent	Good	Poor

From Gordon SJ, Chatzinoff M, Peikin SR: Medical care of the surgical patient with gastrointestinal disease. Med Clin North Am 71:433, 1987; with permission.

mortality.[4, 8] Surgery in patients with Child's class A usually can be performed without significant complications, whereas class B and C cirrhotic patients are at increased risk of morbidity and mortality and require careful preoperative preparation.

Coagulopathies must be treated preoperatively. Vitamin K administration, 10 mg IM or 5 mg IV, should correct vitamin K deficiency in 24 to 48 hours. If this fails to correct the disorder, indicating intrinsic synthetic dysfunction, or if there is insufficient time to allow for correction with vitamin K, then treatment with fresh frozen plasma for clotting factors and cryoprecipitate for fibrinogen must be considered. The platelet count should be raised to a level of $100{,}000/\text{mm}^3$.

Maintenance of adequate hydration and urine output is essential, particularly when renal dysfunction accompanies liver disease. Hepatorenal syndrome is believed to result from insufficient renal perfusion in patients with severe cirrhosis. Treatment includes ensuring an adequate preload to maintain cardiac output and administration of diuretics and low dose dopamine for the latter's renal vasodilating and antialdosterone effects. Volume management is best guided by central venous or pulmonary capillary wedge pressures.

Hypoglycemia and electrolyte disturbances should be corrected. Hepatic encephalopathy with elevated ammonia levels may require treatment with neomycin, lactulose, and protein restriction.

When choosing anesthetic agents one must consider the effects of altered hepatic function on the pharmacokinetics of the drugs to be used as well as the effects of the drugs on liver function. Both regional and general anesthesia can decrease hepatic blood flow 20% to 30%.[5, 13] An anesthetic-induced fall in hepatic oxygen uptake may not be well-tolerated in patients with pre-existing liver disease. Regional anesthesia may be contraindicated in the presence of coagulopathy. Drugs implicated in hepatic dysfunction, such as halothane, are best avoided. The alcoholic may have an increased tolerance to anesthetic agents because of hepatic enzyme induction and central nervous system tolerance. Decreased drug metabolism by the liver leads to drug accumulation and a prolonged half-life. Reduced plasma cholinesterase interferes with the metabolism of succinylcholine and estertype local anesthetics. For albumin-bound drugs (e.g., barbiturates), decreased protein binding and an increased free fraction may increase sensitivity to drug effects. For gamma globulin–bound drugs (e.g., pancuronium), increased protein binding may necessitate an increased initial dosage to produce a desired effect. Opioid administration raises common bile duct pressure and may induce biliary colic. True opioid-induced sphincter of Oddi spasm may confound the results of intraoperative cholangiogram, but this occurs in only 3% of such cases.[12]

For patients with end-stage liver disease, orthotopic liver transplantation is the only curative therapy available. The urgency of the procedure may limit the time available to correct the numerous abnormalities present in the patient with hepatic failure. The anesthesiologist must prepare for large fluid shifts, rapid blood loss, coagulopathies, and dramatic hemodynamic changes. Several large-bore venous catheters must be inserted. Continuous arterial and cardiac filling pressures are monitored. Because the inferior vena cava and occasionally the abdominal aorta are cross-clamped intraoperatively, arterial and venous access catheters are placed above the diaphragm. Massive blood loss requires the use of cell-saving devices, and blood products must be readily available. Hypothermia often develops but can be limited by warming the room, the fluids, and inhaled gases, placing reflective drapes, and using a warming blanket. Because ascites may raise intra-abdominal pressure, aspiration precautions should be taken and rapid sequence induction performed.

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Preanesthetic Assessment of the Patient with Coagulopathies

*Jill Fong, MD**

The hemostatic mechanism is designed to maintain blood within injured blood vessels. When vascular integrity is lost, bleeding occurs. There are four components of the hemostatic mechanism: vascular reaction, platelet function, coagulation cascade, and clot lysis. In response to a vascular injury, all four components function simultaneously. If any of these components are abnormal, persistent bleeding or excessive thrombosis can occur.

NORMAL HEMOSTATIC MECHANISM

Vascular Integrity

Vascular integrity is required to prevent bleeding. In an attempt to maintain vascular integrity, the human body has strengthened those vessels that experience either high pressure or frequent trauma. An interruption of vascular integrity initiates the hemostatic mechanism. Blood will escape from an injured vessel as long as the pressure within the vessel exceeds the orifice pressure. To minimize blood loss, an injured vessel undergoes reflex vasoconstriction in an attempt to shunt blood away from the damaged region. Large breaks in vessel walls, especially those under high pressure, cannot be sealed by platelets or fibrin meshwork. Bleeding will persist until vascular integrity is restored. Platelets and the coagulation cascade stop bleeding if the damage to the blood vessel is small, vascular constriction has occurred, and intravascular pressure does not displace the platelet plug.[5]

Platelets

Platelets are crucial to initiating and localizing clot formation. Platelets are blood elements that are released from megakaryocytes

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in bone marrow and have a half-life of 7 to 10 days in the healthy human being. Platelets circulate, making themselves immediately available to form a cohesive, tightly aggregated mass, the platelet plug. It represents a complete, although short-acting, hemostatic unit.

Once vascular integrity has been disrupted and subendothelial tissue exposed, platelets serve two functions. They form a temporary plug that seals the vascular injury, and they also provide a platelet phospholipid, platelet factor 3 (PF-3), on which the coagulation cascade proteins become activated.

When platelets come into contact with a damaged vessel, they become activated and adhere to the subendothelial connective tissue, spread over the damaged surface, release multiple compounds from their storage granules, and aggregate with other platelets to form the platelet plug. One of the granule compounds released is ADP, which signals other platelets to aggregate and also causes vasoconstriction. Therefore, the amount of ADP released controls aggregation. Locally released prostaglandins regulate ADP release by the second messenger, cyclic adenosine monophosphate (cAMP). The prostaglandin, thromboxane A_2, is synthesized by activated platelets from arachidonic acid with the aid of cyclooxygenase. Thromboxane A_2 decreases cAMP levels, which increases ADP release, and thus, platelet aggregation. Conversely, prostacyclin (PGI_2) is synthesized by endothelial cells from arachidonic acid. PGI_2 increases cAMP levels, leading to decreased ADP release and platelet aggregation. Therefore, localization of platelet aggregation occurs naturally. PGI_2 prevents platelet aggregation from extending past the injured area while thromboxane A_2 leads to platelet aggregation of the area of injury. Platelet aggregation normally takes place within seconds of vascular injury.

The formation of the unstable platelet plug results in the exposure of the phospholipid PF-3 on the platelet surface membrane. PF-3 functions as a congregating place for the proteins of the coagulation cascade. Fibrin, the end-product of the coagulation cascade, stabilizes the platelet plug.[1, 5]

Coagulation Cascade

The coagulation cascade is an enzymatic cascade with sequential activation of a series of inactive coagulation proteins. The factors in the coagulation cascade are listed in Table 1. The Roman numerals were assigned in order of discovery and, therefore, imply nothing about the sequence of action. Most of the circulating coagulation factors are cleavage enzymes, serine proteases. Each of these proteases circulates as an inactive enzyme precursor. Activated serine proteases are designated by adding an "a" to their Roman numeral designation. Each activated factor only activates the factor immediately following it in the coagulation cascade. The other factors that are not serine proteases are factors III, IV, V, VIII, and fibrinogen. Factors V and VIII are cofactors that circulate only in the active form and are very labile. The coagulation factors circulate in inactive forms in high concentrations. The rate limiting step of the coagulation cascade is the

Table 1. *Factors of the Coagulation Cascade*[5,22]

ROMAN NUMERAL	COAGULATION FACTOR	MINIMUM LEVEL FOR SURGICAL HEMOSTASIS (% normal)	IN VIVO HALF-LIFE
I	Fibrinogen	50–100	4–6 d
II*	Prothrombin	20–40	3–4 d
III	Platelet factor 3 (thromboplastin)		
IV	Calcium		
V	Proaccelerin (labile factor)	5–20	15–24 h
VII*	Proconvertin (stable factor)	10–20	4–6 h
VIII	Antihemophiliac factor A	25–30	8–12 h
IX*	Antihemophiliac factor B (Christmas factor)	20–25	18–30 h
X*	Stuart-Prower factor	10–20	40–60 h
XI*	Antihemophiliac factor C	20–30	45–60 h
XII*	Hageman factor	0	50–70 h
XIII	Fibrin stabilizing factor	1–3	3–5 d

* Serine proteases.

availability of an appropriate phospholipid surface, i.e., PF3 or tissue phospholipid, on which to interact. Aggregated platelets and injured tissue cells provide these substrates.

The reactions leading to fibrin formation are divided into two overlapping pathways, the intrinsic and extrinsic pathways. The final portion of both pathways is the same: the formation of a fibrin clot from fibrinogen (Fig. 1).

The intrinsic coagulation cascade begins intravascularly with the activation of factor XII. Factor XII is activated by surface contact with a denuded vessel to become XIIa, which then activates factor XI, and the next step takes place on the activated platelet surface, PF-3.

The extrinsic pathway serves as an alternative pathway to activate factor X and the fibrin clot. A tissue phospholipid binds calcium and factor VII, thus leading to the activation of factor X.

The final common pathway begins with the activation of factor X and continues to the final insoluble fibrin clot. Thrombin, factor IIa, catalyzes the conversion of soluble fibrinogen to fibrin monomers and activates factor XIII, which catalyzes the formation of irreversible covalent bonds between adjacent fibrin monomers. This changes the fibrin clot from an easily dissociated compound to a highly cross-linked structure holding the platelet plug in place.

Regardless of the phospholipid, calcium is required to bind the serine proteases to the phospholipids by an electrostatic charge. Vitamin K enzymatically adds a gamma carboxyl group to factors II, VII, IX and X, the vitamin K–dependent factors, which prepares them to bind to platelet or tissue phospholipid electrostatically. If the vitamin K–dependent factors are not carboxylated, they cannot become activated subsequently.

Once the coagulation factors are active, their action is localized

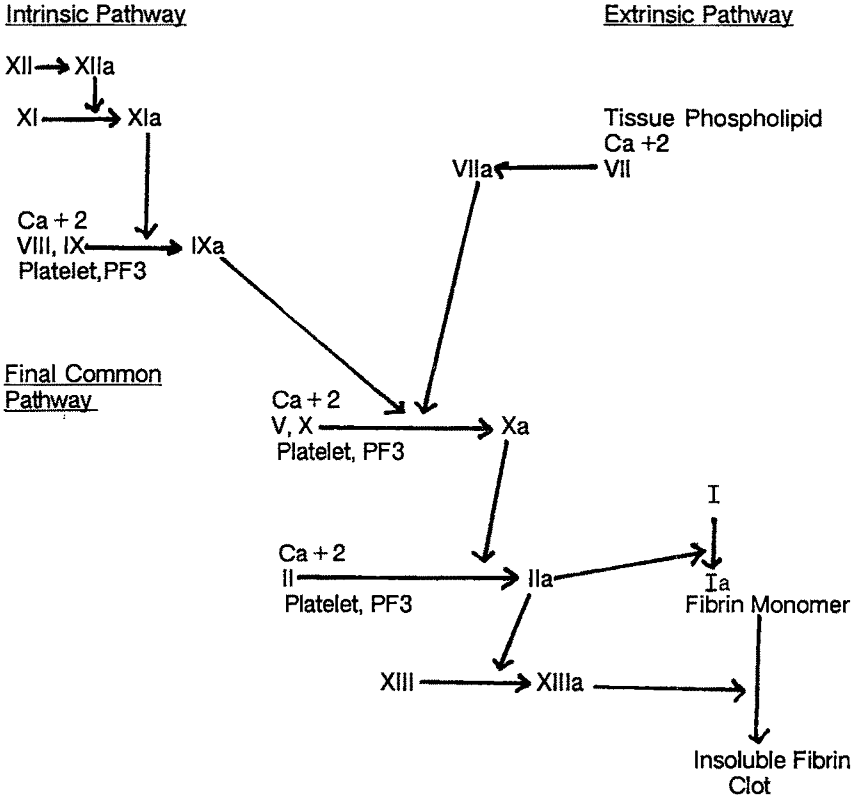

Figure 1. The coagulation cascade.

to the site of injury by three major mechanisms: (1) dilution of activated factors by rapid blood flow, (2) removal of activated factors by the reticuloendothelial system, and (3) neutralization of activated factors by circulating anticoagulants, antithrombin 3 (AT-3), protein C, α_2-macroglobulin, α_1-antitrypsin, and C1-esterase inhibitor. AT-3 is a polypeptide produced in the liver that can bind and inactivate the serine proteases, IIa, IXa, Xa, XIa, and XIIa; factor VIIa appears to be relatively resistant to AT-3. Normally, this binding occurs slowly but heparin binds to AT-3 causing a conformational change that accelerates AT-3's binding of the active serine proteases. Protein C is a serine protease that is synthesized in the liver and dependent on Vitamin K. Once activated, protein C destroys factors V and VIII and initiates clot lysis by releasing tissue plasminogen activator.[3, 5, 21]

Clot Lysis

To reestablish normal circulation, the fibrin clot eventually must be removed. Lysis must proceed slowly, as too much too soon may result in rebleeding from an incompletely healed vessel. The lytic system is similar to the coagulation cascade in that it is activated by factor XIIa intravascularly or by tissue activators extravascularly. In either case, plasminogen is activated to plasmin. Plasmin cleaves both fibrinogen and fibrin into small fragments, effectively lysing the fibrin clot. These small peptides are called fibrin split products (FSP) or fibrin degradation products (FDP). FSPs are removed from the circulation by the reticuloendothelial system, especially the liver's Kupffer cells. If they are not removed and reach high concentrations, the FSPs can inhibit clot formation by preventing the cross-linking of fibrin monomer and causing platelet dysfunction.

Clot lysis is localized by two mechanisms. Factor XIIa or tissue activators are required to convert plasminogen to plasmin and plasmin is rapidly inactivated by antiplasmin that circulates in the blood. Because plasmin is inactivated rapidly by circulating antiplasmin, physiologic fibrinolysis occurs within the clot. The plasminogen initially incorporated into the fibrin clot during formation is converted to plasmin causing clot lysis from the inside out.[5]

EVALUATION OF THE HEMOSTATIC MECHANISM

History

It is important to question patients about possible coagulation abnormalities. One of the best ways to detect such an abnormality is to ask questions about the hemostatic response to prior surgery. Abnormalities also may manifest as frequent and profuse nose bleeds, easy bruising, bleeding gums with normal brushing of teeth, family history, and so forth. In addition, the medications being taken should be reported. Any history suggesting coagulation problems should warrant further investigation of the preoperative patient.

Physical Examination

On physical examination, petechiae can signal a coagulation defect involving platelets or vascular integrity. Ecchymosis or joint hemarthrosis are more likely to be seen with disorders of the coagulation cascade.

Laboratory Tests

Table 2 lists the laboratory tests of coagulation. The mainstays of platelet testing are the platelet count and bleeding time. The platelet count tests the quantity of platelets. An estimate of the platelet count can be obtained from a peripheral blood smear; 10 to 15 platelets per high power microscopic field is normal. Precision determination of platelet number is done by phase contrast microscopy hemocytometer counts. The minimum number of platelets required to prevent surgical bleeding provided there are no other platelet abnormalities is 50,000 platelets per mm^3.

Bleeding time measures both platelet quality and quantity. If the

Table 2. *Tests of Coagulation*[5,25]

TEST	NORMAL VALUE
Platelet count	150,000–400,000 mm^3
Bleeding time	3–7 min
Partial thromboplastin time (PTT)	60–85 s
Activated PTT	25–35 s
Prothrombin time (PT)	11–14 s
Thrombin time (TT)	13–40 s
Reptilase time (RT)	14–21 s
Prothrombin-proconvertin time (P & P)	11 s or 60% control
Fibrinogen level	200–400 mg/dL
Activated clotting time (celite)	<120 s
Whole blood clotting time (glass tube)	2.5–4.25 min
Fibrin split products	<10 μg/mL

platelets are qualitatively normal, the bleeding time is normal when greater than 100,000 platelets per mm^3 are present. When the platelet count is between 10,000 and 100,000 platelets per mm^3, the bleeding time is linearly related to platelet number by the following equation: Bleeding time (minutes) = 30.5 − (platelet count/3850). If the platelets are quantitatively normal but qualitatively abnormal, the bleeding time also will be abnormal. A bleeding time is performed by inflating a blood pressure cuff to 40 mm Hg, making a standard incision 1 mm deep and 9 mm long on the flexor aspect of the arm, and blotting the wound every 30 seconds until no serum is blotted. The bleeding time is the time from incision to absence of serum.

In the presence of a normal platelet count but abnormal function, tests such as platelet aggregation studies can be done to pinpoint the reason for this functional abnormality.[1, 5, 22]

Tests of the Coagulation Cascade

There are a variety of tests that evaluate the coagulation cascade. In testing this there are two basic requirements. First, all tests require the conversion of fibrinogen (I) to fibrin (Ia) to provide an endpoint for the test. The fibrin clot can be examined visually or detected using light beams, magnets, photoelectric cells, and so forth. Second, all of the collecting tubes must have a calcium chelating agent to prevent the specimen from clotting before it arrives at the laboratory. Therefore, calcium must be re-added to the specimen for many of the tests.

Two common tests are the prothrombin time (PT) and partial thromboplastin time (PTT), which evaluate the extrinsic and intrinsic pathways, respectively. Neither test measures factor XIII however, and both lack sensitivity because 60% to 80% activity loss of the various coagulation factors is required to prolong these tests. Because these patients are still at increased risk from surgical bleeding, a complete history is important.

By adding tissue phospholipid, platelets, and calcium to citrated plasma, the extrinsic pathway is tested. The test is called a PT. The PT is prolonged when factors VII, X, V, II, or I are deficient or when heparin or coumarin are present.

To test the intrinsic pathway, a PTT can be done by adding a platelet phospholipid-like material and calcium to citrated plasma and timing until the specimen clots. If factors XII and XI are surface activated before the addition of platelet phospholipid and calcium, the test is an activated PTT (aPTT), which has been shown to be more reproducible than the PTT. The PTT may be prolonged when factors XII, XI, VIII, IX, V, X, II, or I are deficient or when FSP or heparin are present.

The thrombin time (TT) checks the conversion of fibrinogen to fibrin by adding thrombin (IIa) to the citrated plasma. The TT is abnormal when factor I is low or abnormal or in the presence of heparin or FSP.

Fibrinogen concentration is determined by adding large quantities of factor IIa to citrated plasma and comparing the specimen's clotting time with those of known fibrinogen levels.

If the PTT or PT is abnormal, the deficient factor can be determined by mixing studies. Plasma that is deficient in each of these factors can be mixed with the patient's plasma. The plasma that does not correct the PT or PTT identifies the deficient factor.

Reptilase time is a modification of the TT. Reptilase, unlike thrombin, is not affected by heparin. The reagent, reptilase, cleaves fibrinogen into fragments that cross-link even in the presence of FSP. Thus, a reptilase time is normal when the TT is abnormal because of heparin or FSPs.

A prothrombin-proconvertin time is a variation of the PT. Factors V and I are added to citrated plasma, calcium, platelets, and tissue phospholipid. The specimen is then diluted with normal saline. Approximately 10% to 20% of the normal concentration of each coagulation factor is required for normal coagulation. Therefore, in the presence of heparin, diluting the specimen decreases the heparin concentration and leads to the normalization of the time value. When factors II, VII, and X are deficient, this test is abnormal.[5, 21, 22]

Tests of Platelets and the Coagulation Cascade

To test both platelets and the intrinsic pathway, an activated coagulation time (ACT) or whole blood clotting time (WBCT) time can be done. The ACT is performed by adding celite to a whole blood specimen and determining the time required to clot formation. WBCT is done by placing whole blood in a glass test tube and visually observing clot formation.[5]

Tests of the Fibrinolytic System

Clot lysis is detected by the presence of fibrin split products. The amount of FSPs is determined quantitatively by mixing plasma with antibodies against these degradation products. Serial dilutions are made until no visible clumping is seen. The level of FSP is abnormal and increased with active fibrinolysis.[5]

Thromboelastogram

The thromboelastogram determines the adequacy of the hemostatic mechanism by evaluating platelet number and function, the coagulation cascade, and clot lysis. Whole blood is placed into a rotating metal cuvette in which a piston is suspended. As the hemostatic mechanism proceeds, the adequacy of the clotting and lysing mechanism is determined by measuring and recording the shear elasticity of the fibrin strands formed from the cuvette to the piston.[22,23]

COMMON DISORDERS OF THE HEMOSTATIC MECHANISM

Disorders of Vascular Integrity

There are basically two categories of vascular integrity disorders. The first consists of normal vessels subjected to trauma such as surgery and the second of abnormal vessels bleeding despite an intact clotting system. For instance, in patients taking corticosteroids or who have lost connective tissue support, the vascular response to injury such as vasoconstriction and shunting is impaired and, as a result, bleeding can occur.[21]

Platelet Disorders

Both quantitative and qualitative platelet abnormalities can cause bleeding. Quantitative abnormalities can occur from abnormal platelet production, decreased survival, abnormal distribution, or increased platelet loss. Decreased platelet production can occur if the bone marrow's megakaryocyte number is reduced. This can occur if the bone marrow is infiltrated with tumor or has been destroyed by radiation or drugs such as ethanol, chemotherapeutic agents, estrogen, and thiazides. Increased platelet production also can occur. Thrombocytosis may be a physiologic response to an underlying condition or may be idiopathic such as in polycythemia vera. The latter may be associated with abnormal platelet function. Platelet destruction and removal can have an immunologic or nonimmunologic basis. For instance, in idiopathic thrombocytopenic purpura (ITP), platelet survival is decreased because of immune-mediated platelet destruction; IgG antiplatelet antibodies attach to platelets and enhance their destruction in the reticuloendothelial system. Patients who have disseminated intravascular coagulopathy (DIC) or artificial heart valves exhibit problems of decreased platelet survival. Hypersplenism due to a variety of causes can result in excessive platelet sequestration and a decrease in the number of circulating platelets.[5, 21, 22] Massive transfusions with platelet-poor bank blood or crystalloid lead to a decrease in platelet number because of platelet washout.[14]

Qualitative platelet abnormalities, as exhibited by an abnormal bleeding time with normal platelet number, may be inherited or acquired. Inherited platelet abnormalities may be due to congenital lesion in any one of the five steps in platelet mechanics: (1) contact, (2) adhesion, (3) spreading, (4) release, or (5) aggregation. The acquired abnormalities can occur secondary to drugs, uremic plasma, fibrin split products, cirrhotic liver disease, platelet storage, and so forth.

Drugs that can cause platelet dysfunction include aspirin, dextran, aminoglycosides, protamine, nitroprusside, nitroglycerin, and the inhalation and local anesthetics. Aspirin irreversibly acetylates the enzyme cyclooxygenase, preventing the production of the prostaglandin, thromboxane A_2. Thromboxane A_2 is required for the initiation of platelet aggregation. Therefore, platelet aggregation is inhibited and is abnormal for the life of the affected platelet, which is 7 to 10 days. However, these abnormal platelets can aggregate when exposed to ADP released from transfused platelets.[3, 5, 21, 22]

The inhalation anesthetic agents in therapeutic concentration can decrease in vitro platelet aggregation and, therefore, act as anticoagulants.[4, 24] In vivo studies, with nitrous oxide, enflurane, isoflurane, and fentanyl, however, do not support this in vitro data. Halothane is the only agent that prolongs bleeding time. The clinical significance of this may be significant in patients with abnormal platelet number or function.[6, 7] One possible reason for the platelet dysfunction caused by those agents is their ability to decrease platelet membrane permeability and hinder the release of endogenous ADP.

Local anesthetics also inhibit platelet aggregation in vitro. However, this only occurs at very high, toxic, plasma levels and is, therefore, not clinically relevant.[8]

In uremic failure, platelet dysfunction can occur from the accumulation of dialyzable compounds. This dysfunction is often reversible with hemodialysis.[22]

Platelet dysfunction after storage is due to a depletion of energy stores, specifically ATP. The rate of ADP production and thus platelet aggregation is reduced until these energy stores are replenished. Depending upon the temperature and duration of storage, platelet defects occur that can last 8 to 20 hours after transfusion.[1, 5, 21]

von Willebrand's Disease

von Willebrand's disease is usually transmitted by an autosomal dominant mechanism. It is most likely caused by a deficiency or abnormality of a protein, von Willebrand factor, which is important for platelet and factor VIII activity. Factor VIII and von Willebrand factor circulate in normal plasma as a complex protein, factor VIII-von Willebrand protein. There are different variants of von Willebrand's disease. Classically, patients with von Willebrand's disease have a prolonged bleeding time with normal platelet count, low factor VIII activity with variable PTT, and deficient von Willebrand factor. Correction can be accomplished with desmopressin acetate or factor VIII from either fresh frozen plasma or cryoprecipitates.[11,15]

Disorder of Coagulation Cascades

Disorders of the coagulation cascade can be divided into two categories: disorders of inactive or active factors. Inactive factor disorders can be divided into hereditary or acquired disorders. Inactive factor disorders caused by hereditary factors usually involve a single factor and present as a lifelong problem, whereas acquired disorders usually involve multiple factors and present as a sudden onset of bleeding.

The most common hereditary deficiencies of inactive factors are the hemophilias. Hemophilia A is a factor VIII deficiency with a 1:25,000 incidence. Hemophilia B is a factor IX deficiency with an incidence of 1:100,000, and hemophilia C is a factor XI deficiency occurring in 1:1,000,000. All of these disorders cause an abnormal PTT. Specific factor assays have to be done to distinguish between them. Hemophilia A is by far the most common and is transmitted as a sex-linked recessive trait. To prevent spontaneous bleeding, more than a 5% level of factor VIII activity is required. Major surgery requires at least 30% activity, preoperative and postoperative for 2 to 3 weeks. Preoperatively, the patient should be given factor VIII to achieve a 70% to 100% level. Because the half-life of factor VIII is 8 to 12 hours, more must be transfused every 8 to 12 hours for the first 2 to 3 postoperative weeks. Factor VIII can be replaced with cryoprecipitate, which contains 5 to 10 U/mL of factor VIII, or antihemophiliac factor (AHF) concentrate, which contains up to 40 U/mL of factor VIII. Whenever transfusions of human blood products are given, hepatitis or acquired immunodeficiency syndrome (AIDS) may be contracted. The risk of acquiring these infections is higher when pooled donor products, such as cryoprecipitate and AHF, are given. Danazol has been shown to increase factor VIII activity in some patients and may also be used. Similarly, 1-desamino-8-d-arginine vasopressin (DDAVP) has been used successfully to treat some patients with mild hemophilia A.[5, 9–11]

Acquired inactive factor disorders include those caused by antibodies to a specific factor, decreased production from hepatic disease or vitamin K deficiency, or consumption/dilution problems. Vitamin K is a fat-soluble vitamin. A deficiency or ineffectiveness can occur secondary to malabsorption, antibiotic destruction of vitamin K producing intestinal flora, or coumarin. Without vitamin K, four inactive factors, II, VII, IX, and X, cannot bind to the phospholipids. Therefore, they become ineffective factors.[5, 22]

Hepatic disease causes disorders because the liver produces all of the coagulation cascade and fibrinolytic proteins except factor VIII. Liver disease may, therefore, lead to a deficiency of these proteins. Advanced liver disease also may be associated with thrombocytopenia secondary to hypersplenism and disseminated intravascular coagulation (DIC). DIC occurs because the liver is unable to destroy activated clotting factors in the plasma. There is no specific treatment for the hemostatic defect seen with liver disease; basically, treatment is supportive.[21]

Active factor disorders occur when patients receive heparin or have high concentrations of circulating FSPs. Heparin causes AT-3 to bind active serine proteases more avidly. Therefore, heparin affects every factor in the coagulation cascade except factors VIII, V, and I. As a rule, TT is the most sensitive assay for the presence of heparin, followed by the aPTT and ACT. The PT is the least sensitive assay. Protamine can antagonize the effects of heparin by competing with AT-3 for the available heparin. The protamine-heparin complex is devoid of anticoagulant activity. FSPs in high concentration prevent the cross-linking of fibrin monomers and, therefore, prevent the formation of a stable clot.[5]

Disorders of Clot Lysis

Abnormal fibrinolysis and normal but increased fibrinolysis can occur. Abnormal fibrinolysis occurs after the administration of urokinase or streptokinase. Both of these agents are tissue activitators that cause plasminogen to be converted to plasmin. They are used to dissolve thromboemboli in the pulmonary, cardiac, or venous circulation. However, the circulatory plasmin can cause bleeding because it lyses not only unwanted clots but all clots. Similarly, urologic (specifically prostatic), surgery may result in the release of large amounts of urokinase. Epsilon aminocaproic acid (EACA) prevents plasmin formation and may be indicated when abnormal lysis occurs without active clotting occurring. This therapy should be guided by a hematologist.[2,5] Normal but increased fibrinolysis occurs in patients with disseminated intravascular coagulation.

DIC

DIC always occurs secondary to some other initiating disease state. This disorder occurs when the normally localized hemostatic process becomes uncontrollable and there is widespread clotting followed by diffuse lysing of this clotting. It is a consumptive coagulopathy. Factors V, VIII, and I are used once and destroyed, as are platelets; thus, these factors are consumed during the clotting process. The active serine proteases can catalyze multiple reactions. Although the primary problem in DIC is excessive clotting, bleeding usually occurs because of the widespread consumption of factors and clot lysis. Occasionally only thrombotic stigmata are present.

Causes of DIC include (1) disorders that present with long regions of denuded vascular endothelium such as burns and vasculitis, (2) the presence of intravascular immune complexes or bacteria such as occurs with sepsis, and (3) disorders associated with large amounts of intravascular tissue phospholipids such as those that occur with massive crush injuries, necrotic malignant tumor tissue, amniotic fluid embolus, retained fetal tissue, and hemolytic transfusion reactions.

Laboratory values show that the PT, PTT, and TT are prolonged. The platelet number and fibrinogen levels are low. The FSPs are increased, which produces platelet dysfunction and inhibits fibrin cross-linking. Additionally, anemia may be present secondary to the fragmentation of red blood cells, as they traverse fibrin-filled capillaries.

The treatment of DIC is to resolve the underlying cause. Treating the coagulation disturbances is only a temporary solution. The temporary treatment of DIC consists of "fueling the fire" by giving blood products as necessary to minimize bleeding. In a very select group of patients, heparin may be used temporarily to neutralize the activated coagulation factors and prevent further clotting and consumption. There is the hazard, however, of bleeding from sites requiring clotting. This therapy requires the guidance of a hematologist. Survival depends upon the resolution of the initiating problem.[5, 12, 21, 22]

GOALS OF ANESTHETIC MANAGEMENT

It is important to obtain a detailed history and physical examination preoperatively. This information will dictate the selection of the appropriate coagulation studies. Once the cause of the coagulopathy is pinpointed, therapy can be instituted as needed. The old concept of giving whole blood for everything has been replaced by component therapy. In this way each patient is only exposed to the required blood component.[16, 17]

Regional anesthetic techniques are relatively contraindicated in patients with coagulation abnormalities. An area of controversy is the use of regional anesthesia in patients who are currently on or soon to be anticoagulated. Owens et al[19] reported spinal hematomas in 33 patients after a lumbar puncture or spinal anesthesia. These patients were anticoagulated or thrombocytopenic. Rao and El-Etr[20] reported on 3164 patients receiving continuous epidural blocks and 847 patients receiving continuous spinal anesthetics. About 1 hour after induction of anesthesia, all of these patients were anticoagulated with heparin to increase their ACT-2 times baseline. None of these patients developed epidural or subarachnoid hematomas.[20] Similarly, 40 cardiac patients received intrathecal morphine before heparinization without incident.[13] Odoom and Sih[18] found no adverse outcomes in anticoagulated patients receiving epidural anesthesia. They concluded that epidurals are safe if the ACT is less than 200 seconds and analgesia is allowed to wear off between each reinforcement so that motor and sensory function can be assessed. Collectively, the data show that spinal and epidural hematomas can occur spontaneously in association with anticoagulation regardless of anesthetic intervention. Therefore, in the anticoagulated patient population, each situation should be evaluated on an individual basis and the benefits carefully weighed against the risks of a regional anesthetic.

Finally, general anesthesia is often the anesthetic of choice in patients with coagulopathies. Any general anesthetic technique can be used. In patients with platelet abnormalities, however, it may be wise to avoid halothane.[6]

SUMMARY

In evaluating the perioperative patient with coagulopathies, a detailed history and physical examination aid in the appropriate selection of in vivo and in vitro coagulation studies. The hemostatic mechanism is composed of four components, vascular reaction, platelet function, coagulation cascade, and clot lysis. Once vascular integrity is lost, bleeding occurs. Normally, these four components function simultaneously to provide rapid and localized hemostasis. Injured blood vessels reduce their blood flow by vasoconstriction, shunting, and tissue swelling. The exposed subendothelial vascular lining allows platelets to adhere to and plug the vascular injury. The platelet plug itself only temporarily stops bleeding. The platelet surface initiates and supports the enzymatic reactions of the coagulation cascade. This cascade reinforces the platelet plug with a fibrin meshwork that leads to a stable clot. To reestablish normal blood flow in a previously injured vessel, clot lysis is required to remove the fibrin clot and any trapped platelets. Abnormalities of the hemostatic mechanism can lead to persistent bleeding or excessive thrombosis. Once the reason for a coagulopathy is discovered, the appropriate therapy can be instituted.

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Preanesthetic Assessment of the Obstetric Patient

*Steven S. Schwalbe, MD**

GENERAL CONSIDERATIONS

The preoperative evaluation of the obstetric patient begins in the same fashion as that for any patient undergoing anesthesia. History, physical examination, and appropriate laboratory values are scrutinized within the context of the contemplated procedure. Such considerations may first appear to be too basic to mention. Nevertheless, it is often these elemental steps that are overlooked in the hectic atmosphere of a busy obstetric anesthesia service.

There are, however, certain aspects unique to the obstetric patient. Considerations regarding the delivery, the well-being of the fetus, and the unique physiologic changes of pregnancy must be taken into account in the preanesthetic evaluation.

The timing of this evaluation varies widely. Ideally, the first contact between the pregnant woman and the anesthesiologist should occur long before the planned delivery date. This not only affords the opportunity to request any necessary consultations or laboratory studies but enables the anesthesiologist to have a frank discussion with the gravida regarding the pros and cons of the available methods of analgesia. Such conversations often suffer when they are held in the delivery room with an anxious, apprehensive parturient who is experiencing severe labor pain.

Unfortunately, the nature of pregnancy is such that this ideal, relaxed scenario is not always possible. Anyone who spends a substantial amount of time in the labor and delivery suite has encountered the experience of performing an evaluation "on the run" between the admitting area and the delivery or cesarean section room. Nevertheless, if an effort is made to see as many women as possible prior to

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admission, the reward can be a more comfortable experience for both the woman and the anesthesiologist.

Many women approach delivery with trepidation. They are often fearful for the safety and well-being of their child and feel a certain loss of control in a situation in which they will encounter obstetricians, anesthesiologists, high-technology intrauterine monitoring, and so forth. Other women are determined to take a very active role in the events surrounding their labor and delivery and may be exceptionally well-informed about obstetric considerations and anesthetic techniques. Unfortunately, the mass media, from which many women gather their information on obstetric procedures, is not always accurate or complete in providing information. Consequently, in approaching the gravida, an anesthesiologist must be both reassuring and patient. Explanations of objectives, advantages, and disadvantages of the various modalities of analgesia should be presented in nontechnical terms and the woman should be made to feel that she is involved in the decision making process as much as possible. The history obtained from a gravida must not be abbreviated. It is a common but mistaken belief that pregnant women are all healthy and free of medical problems. While this is fortunately true for most women, it is by no means universal. The incidence of heart disease is 0.4% to 4.1%.[80] Asthma is common and the symptoms may worsen during pregnancy.[83] Hypertensive disorders, including preeclampsia, involve 5% to 10% of all pregnancies in the United States.[52] Gestational diabetes is not unusual and preexisting diabetes mellitus may be severely affected by pregnancy.[24] Conditions that tend to affect young women, such as systemic lupus erythematosus, are also encountered.[68] In some inner city patient populations as many as 2% of the women delivering test positive for the human immunodeficiency virus (HIV).[49] The author's own service has recently administered to women with pheochromocytoma, sickle cell disease, thalassemia, cerebral arteriovenous malformations, abnormal pseudocholinesterase, osteogenesis imperfecta, cocaine abuse, lymphoma, and a history of malignant hyperthermia, among others.

OBSTETRIC HISTORY

It is important to obtain information about the woman's prior and current obstetric history. Communication with the obstetrician as well as the gravida is essential in formulating an anesthetic management plan. If an operative delivery is planned, the anesthesiologist should be aware of conditions that can lead to heavy blood loss. A low anterior placenta, placenta accreta, abruptio placenta, or placenta previa signal the anesthesiologist to be prepared with large bore intravenous access and ready availability of blood and crystalloids. Such situations may also favor the administration of general anesthesia as opposed to regional to facilitate control of intraoperative blood loss and hypovolemia. Many cases in which a vaginal delivery is planned require the anesthesiologist to stand by and be prepared for immediate intervention or induction of anesthesia should the need arise. Multiple gestations, breech extractions, external version maneuvers, forceps deliveries, or vacuum extractions are examples of this category. Still other circumstances, such as shoulder dystocia or uterine inversion, may be completely unexpected but will require rapid, expert assistance from the anesthesiologist to avoid disaster.

A history of cesarean section or other uterine surgery always raises the possibility of placenta accreta. In this condition the placenta becomes adherent to the myometrium and fails to separate properly at delivery. This in turn can lead to a massive, rapid loss of blood, which may be controllable only by hysterectomy.[20] Placenta accreta occurs most often in the area of a prior surgical scar.[77] With the cesarean section rate in the United States approaching 23%,[81] this condition, once uncommon, is now seen with increasing frequency.[19] With each subsequent cesarean section, the risk of gravid hysterectomy rises dramatically.[3] It is therefore incumbent upon the anesthesiologist to communicate with the obstetrician to determine if the placenta is in the area of an old surgical scar whenever there exists a history of prior uterine surgery.

PHYSIOLOGY OF PREGNANCY

To properly evaluate and be prepared for the obstetric patient, one must be cognizant of the physiologic changes that normally occur with pregnancy. Virtually all systems in the body undergo some alterations that serve the gestation or prepare for the coming labor and delivery. These changes can be either the result of hormonal influences, mechanical changes from the enlarging uterus, or both.

Cardiovascular Changes

Blood volume starts to increase in the first trimester, probably as early as the fifth to eighth week of gestation.[67] The maximum rate of increase occurs in the second trimester. By the 30th week of gestation, blood volume is 80 to 85 mL/kg as opposed to the nonpregnant level of 60 to 65 mL/kg.[16] It then remains fairly constant until delivery. The increased volume perfuses the gravid uterus, enlarging breasts, kidneys, and skin. Most of this increase is due an increase in plasma volume of 45% to 50%.[5] Although red cell mass also increases, the elevation is far smaller, about 20% to 30% above nonpregnant values. As a consequence, although there are more circulating red blood cells, the hematocrit level and blood viscosity are diminished by dilution. Thus, with such a large increase in blood volume, the pregnant woman is well-prepared for the expected loss of 500 mL of blood during a vaginal delivery or the average loss of 900 mL with cesarean section.[70]

Accommodating these changes in blood volume are parallel increases in cardiac output. Both stroke volume and heart rate increase, although increases in stroke volume predominate in early pregnancy.[85] Cardiac output increases further with labor and again in the immediate postpartum period when left ventricular stroke volume may reach 180% of prelabor values.[84] The interval after delivery may therefore have profound consequences for women who cannot tolerate rapid changes in output and blood volume, such as those with mitral stenosis. Blood volume and cardiac output usually have returned to normal by the end of the second postpartum week.[88] Peripheral vascular resistance decreases during pregnancy, thus blood pressure does not change greatly during gestation, despite the increase in output. Central venous pressure remains normal. These changes are the results of hormonal influences.[92]

Toward the end of the second trimester the weight of the gravid uterus becomes great enough to compress the inferior vena cava and the lower abdominal aorta when in the supine position. Obstruction of the vena cava compromises venous return and in 10% to 15% of pregnant women at term results in clinical signs and symptoms such as hypotension, tachycardia, diaphoresis, nausea, vomiting, right lower quadrant pain, and fainting.[43] Many gravidas who are asymptomatic have compensated for this compression by an increase in heart rate, sympathetic tone, peripheral vasoconstriction, and by a rich collateral circulation by the epidural plexus of veins into the azygous system. However, in the presence of a high sympathetic blockade as with a regional anesthetic, these women, too, may show overt signs of diminished cardiac output. Obstruction of the lower aorta may not cause any clinical signs or symptoms in the mother, but uterine blood flow can be severely diminished, resulting in fetal compromise.[53] Total or near total compression of the lower aorta can be demonstrated in 86% of gravidas close to term.[8] There is clearly a high probability of fetal compromise from aortocaval compression without any maternal symptoms if the gravida can compensate for caval obstruction. A pregnant woman must therefore not be allowed to lie in the supine position, irrespective of the presence or absence of maternal signs of compromise. Uterine displacement must be maintained at all times. Tilting the patient 15° to the left or placing a wedge under the right hip is the usual way in which this is achieved. This, however, is not always the optimal position.[75] In questionable cases and particularly in cases of multiple gestation or polyhydramnios, blood pressure measurements in the leg or cardiac output determinations can assist in determining the proper maternal posture.[33]

As the enlarging uterus displaces abdominal contents, the diaphragm is moved cephalad and the heart rotated anteriorly and to the left. As a result, the heart may falsely appear to be enlarged on radiographs and the electrocardiogram of a pregnant woman usually shows left axis deviation.[50] The ECG may also show reversible ST segment or T-wave changes.[62] A small Q wave may be seen, particularly in lead III.[64] Premature atrial contractions and ventricular extrasystoles are common but usually benign. Auscultation often reveals grade I or II systolic flow murmurs or a fourth heart sound (S4).[26] These are usually of no clinical significance and will resolve postpartum. Murmurs louder than grade II, third heart sounds (S3), or diastolic murmurs, however, deserve further investigation.

The white cell count, primarily neutrophils, increases during pregnancy. During the third trimester, a parturient's absolute neutrophil count is almost double what it will be postpartum.[69] The lymphocyte count does not change greatly, however, pregnancy does depress lymphocyte activity and cell-mediated immunity. The gravida may, therefore, be more susceptible to viral infections.[36]

The platelet count diminishes during pregnancy, probably as a dilutional effect from the increased blood volume.[69] Platelet function, however, remains adequate during normal gestation, and increased circulating levels of clotting factors lead to a hypercoagulable state in the normal gestation.[41]

Pulmonary Changes

Changes in ventilatory patterns begin in the first trimester.[1] Tidal volume increases by 40% at the expense of the expiratory reserve, and respiratory rate may increase by 1 to 2 breaths per minute.[65] This increase would yield a 50% increase in minute ventilation and, since dead space is unchanged, a 70% increase in alveolar ventilation (Table 1). Hyperventilation results in a normal arterial partial pressure of carbon dioxide (P_{CO_2}) of 32 mm Hg during pregnancy. A normal pH is maintained by an increased renal excretion of bicarbonate, so the normal serum bicarbonate level becomes 22 mEq/L. The resulting arterial blood gas is, therefore, one that resembles a compensated respiratory alkalosis.[71]

The diaphragm is elevated about 4 cm cephalad by the third trimester. Total lung capacity and vital capacity show little change because of compensatory increases in the anteroposterior and transverse diameters of the chest.[51] However, the residual volume of the lung is diminished by 20%. This decrease in residual volume combined with the drop in the expiratory reserve yields a 20% fall in the functional residual capacity (FRC)[10] (Fig. 1).

Because closing capacity (CC) does not change with pregnancy, the FRC:CC ratio is obviously changed. If this change leads to premature airway closure during tidal ventilation, air trapping, atelec-

Table 1. *Pulmonary Volume Changes in Pregnancy*

VOLUME/CAPACITY	CHANGE
Inspiratory reserve volume	No change or slight increase
Tidal volume	40% increasee
Expiratory reserve volume	20% decrease
Residual volume	20% decrease
Vital capacity (IRV + TV + ERV)	No change
Total lung capacity (IRV + TV + ERV + RV)	No change or slight decrease
Functional residual capacity (ERV + RV)	20% decrease
Closing capacity	No change or slight decrease

Abbreviations: IRV-inspiratory reserve volume, TV-tidal volume, ERV-expiratory reserve volume.

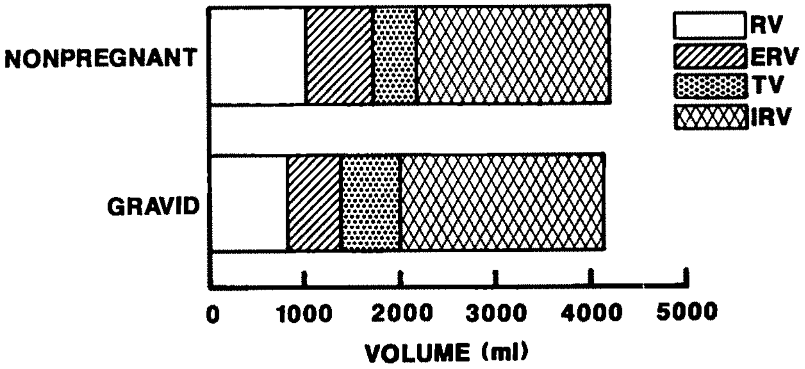

Figure 1. Lung volumes for the nonpregnant woman and the pregnant woman at term. Residual volume = RV, expiratory reserve volume = ERV, tidal volume = TV, inspiratory reserve volume = IRV, and functional residual capacity = FRC. FRC = RV + ERV.

tasis, changes in the alveolar-arterial oxygen gradient, and dyspnea may result. Such a relationship may be particularly important when there are additional conditions superimposed on the pregnancy that also affect this ratio. For example, a history of tobacco smoking or lung disease may increase closing capacity, whereas the presence of obesity, anesthesia, or the lithotomy position may additionally decrease the FRC.

The change in FRC also implies an adverse effect on the pregnant woman's oxygen reserve. When this is combined with her increase in basal oxygen consumption of 25%, it can be readily appreciated that oxygen saturation falls more rapidly in the pregnant than in the nonpregnant woman.[4]

Capillary engorgement of the mucosa occurs throughout the respiratory tract, from the nasal and oropharynx down to the trachea.[10] The lumen of the airway is narrowed and often causes pregnant women to report head colds and nasal stuffiness. Thus, the use of a smaller size endotracheal tube is appropriate. Instrumentation of the nose is avoided, as it can lead to profuse bleeding.

Gastrointestinal Changes

Changes that occur during gestation make the gravida prone to regurgitation and place her at risk for developing an aspiration pneumonitis. The enlarging uterus displaces abdominal contents cephalad, altering the angle between the esophagus and stomach and tending to divide the stomach into antral and fundal pouches.[2] The first change alters the efficiency of the "pinchcock" mechanism of the lower esophageal sphincter in preventing regurgitation; the second hinders gastric emptying. As early as the first trimester, the pregnant woman has an increased gastric pressure and a lower esophageal pressure. In the 45% to 70% of gravidas who experience heartburn and esophagitis this difference is even more exaggerated.[11] Gastric motility is decreased and gastric emptying prolonged as a result of increased progesterone and diminished plasma motilin levels.[14, 18] Investigations have demonstrated that even after an overnight fast, 25% of women scheduled for an elective cesarean section were at risk of aspiration (pH of gastric contents lower than 2.5 and gastric volume greater than 25 mL).[72, 73] Gastric emptying is retarded even more by labor and by the administration of narcotics for analgesia.[60] These alterations in gastric function may become even more prolonged in the postpartum period.[9]

By the 20th week of gestation placental secretions cause increased plasma gastrin levels, elevating the acid, chloride, and enzyme contents of the stomach.[72, 73] Thus, from the end of the first trimester through the first postpartum month, pregnant women must be considered at risk for developing the acid aspiration syndrome, also known as Mendelson pneumonitis[56] (Table 2). Indeed, pulmonary aspiration of stomach contents is a leading cause of maternal morbidity and mortality.[15]

The first step in preventing aspiration is to preserve maternal consciousness and prevent a loss of upper airway reflexes and the ability to clear the airway. The prejudice of obstetric anesthesiologists toward regional anesthesia for operative procedures is, in large measure, rooted in the idea of avoiding this risk of aspiration. Nevertheless, the institution of a regional blockade does not automatically remove the danger. Great care must be taken to avoid both local anesthetic toxicity and the development of hypotension, both of which can lead to depression of the central nervous system. Oversedation with adjuvant medications also must be avoided.

If general anesthesia becomes necessary, measures must be taken to decrease the risk of regurgitation and aspiration. For scheduled, elective cesarean sections, when the time before induction of anesthesia is not a limiting factor, many anesthesiologists administer H_2 histamine receptor antagonists (ie, cimetidine, ranitidine, etc.) orally on the night before surgery and again parenterally 1 to 3 hours before induction of anesthesia.[15] The limitations of this therapy must be understood. While it aids in raising the pH of gastric contents, the

Table 2. *Factors that Increase the Risk of Aspiration in Pregnancy*

- Delayed gastric emptying
  - Increased progesterone
  - Decreased plasma motilin
  - Displacement of the stomach
  - Division of the stomach
  - Labor pain
  - Narcotic administration
- Incompetence of the lower esophageal sphincter
- Elevated gastrin levels
- Increased intragastric pressure

effect is not universal.[91] Aspiration of particulate matter is not affected by these agents. Cimetidine[35] and to a lesser extent ranitidine[37] decrease hepatic blood flow and interfere with liver metabolism of diazepam, propranolol, and lidocaine. Toxic plasma levels of amide local anesthetics may result when cimetidine is used prior to administration of an epidural anesthetic.

Metoclopramide, 10 mg, may be given 15 to 30 minutes before induction to promote gastric emptying.[23, 93] Tone of the lower esophageal sphincter is increased for approximately 40 minutes.[23] The drug is far less effective after narcotic administration.

The administration of a nonparticulate antacid (30 mL of 0.3 mol/L sodium citrate) can rapidly neutralize 255 mL of hydrochloric acid with a pH of 1.0.[38] It is considerably less effective in vivo, probably because of inadequate gastric mixing.[39] Antacid therapy is useful but, like H_2 antagonists, is not effective in all women and offers no protection against aspiration of particulate material. Unlike the antihistamines, it can be given shortly before induction. It is, in fact, generally administered 10 to 30 minutes before the administration of general anesthesia.

The anesthetic induction is also geared toward the full stomach patient. Anticholinergics such as atropine or glycopyrrolate are not administered routinely. They are not effective in decreasing either gastric acidity or gastric volume[72] and can decrease the tone of the lower esophageal sphincter.[23]

Precurarization is controversial. Pretreatment with a small dose of a nondepolarizing muscle relaxant (i.e., d-tubocurarine, 3 mg) can prevent fasciculations and the subsequent rise in intragastric pressure caused by succinylcholine.[57] However, the effects of fasciculations on the tone of the lower esophageal sphincter may actually decrease the difference between the intragastric and gastroesophageal sphincter pressures, the "barrier pressure," and actually be more beneficial in preventing regurgitation, as has been shown to be the case in nonpregnant patients.[78] Precurarization prolongs the onset time of succinylcholine and can lead to premature paralysis.

After adequate preoxygenation with 100% oxygen at nonrebreathing flow rates, the anesthesiologist usually proceeds with a rapid sequence induction with cricoid pressure (Sellick maneuver). In cases involving an expected difficult intubation, an awake, fiberoptic intubation may be attempted. Equipment should be on hand to enable ventilation by a cricothyrotomy if the trachea can not be intubated or the lungs ventilated by mask with cricoid pressure in place. Extubation should only take place in a responsive patient with adequate signs of reversal from neuromuscular blockade.

Renal Changes

Renal blood flow and glomerular filtration rate (GFR) rise rapidly in the first 4 months of gestation until they peak at 150% of nonpregnant levels.[30] Creatinine clearance increases and blood urea nitrogen (BUN) and serum creatinine levels are reduced 40% during a normal pregnancy.[76] This resetting of normal values must be borne in mind when evaluating renal function in states that compromise the kidney, such as preeclampsia, systemic lupus erythematosus, and diabetes. A BUN value greater than 12 mg/dL must be investigated.

Glucosuria is not uncommon, as the renal threshold changes and the increase in GFR may overwhelm the ability of the tubules to reabsorb. Proteinuria of up to 300 mg/day is not uncommon and does not necessarily imply a pathologic condition.[82]

Kidney size increases and renal calyces, pelves, and ureters dilate after the third month of pregnancy. These changes are probably the result of both hormonal and mechanical influences and do not represent a pathologic condition.[87] The enlarged volume of urine in the pelves and ureters and the stasis that occurs partly from mechanical obstruction of the ureters may contribute to the frequency of urinary tract infection during pregnancy.[42]

Central Nervous System Changes

Central nervous system changes of note to the anesthesiologist center around the diminished requirements for both general and regional anesthesia. The amount of local anesthetic needed for spinal and epidural anesthesia is one-third less than for the nonpregnant woman. Traditionally this has been explained by pointing out that the epidural veins are engorged, making the epidural space smaller, and by the increased pressure in the subarachnoid space during labor.[12] There is now evidence, however, that the nerves themselves, under the influence of progesterone, are more sensitive to the effects of the local anesthetics.[27] Increased sensitivity begins in the first trimester before mechanical factors are likely to play an important role.[34] Postpartum, the response of the nerves rapidly returns to normal, and by 36 hours after delivery the dose requirements are essentially the same as in the nonpregnant state.[13]

The requirements for general anesthesia are decreased by 25% to 40% during pregnancy.[63] This change, combined with hyperventilation and a reduced FRC, can lead to an unexpected induction in situations in which simple mask analgesia is intended, an obvious hazard in a woman who is considered to be at risk for aspiration. Vigilance is especially required when this technique is contemplated.

SPECIAL SITUATIONS

Breech Presentation

The breech presentation of the fetus is associated with a wide range of problems. The baby may experience trauma and spinal cord damage, intracranial hemorrhage, and hypoxia and the delivery may be premature. Difficulties with delivery include umbilical cord prolapse, placenta previa, and abruptio placentae.[46] The high rates of fetal morbidity and mortality have resulted in the cesarean section becoming the preferred method of delivery for most of these infants.[21] However, in selected cases, depending on fetal size, position, gestational age, pelvic adequacy, and other maternal and fetal considerations, many obstetricians choose to deliver these babies vaginally.

If the parturient's cooperation is desired, lumbar epidural analgesia can provide excellent analgesia for both labor and the delivery as well as maximal perineal relaxation for delivery of the head. General anesthesia should not be used routinely, as it introduces significant maternal risk.[25] It may become necessary, however, to induce general anesthesia rapidly in the event that the aftercoming head becomes trapped. Therefore, the anesthesiologist must be prepared, having checked all equipment and administered aspiration prophylaxis to the woman.

General anesthesia is useful in the case of the entrapped head primarily because it can provide rapid and profound perineal relaxation. While it is true that the halogenated inhalational agents (i.e., halothane, enflurane, and isoflurane) relax the body of the uterus and the lower uterine segment, they do not dilate the cervix.[58, 59]

Whatever anesthetic method is chosen, a high inspired fraction of oxygen (FIO_2) should be administered to the mother in an effort to minimize fetal hypoxia.

If an abdominal delivery is planned, either general or regional anesthesia may be used for the delivery, and the choice is usually made based on the medical and obstetric considerations at hand other than the fact of a breech presentation.

Tocolytic Therapy

Tocolytics are often administered to inhibit premature labor. On occasion, they may be used to manage an acute fetal distress while preparations are being made for delivery. If tocolysis fails or if the intrauterine conditions are such that the child must be delivered, the anesthesiologist may be faced with the situation of administering anesthesia for an abdominal or vaginal delivery in the presence of these agents.

Uterine relaxation is an effect of β-2 receptor activation. Ritodrine and terbutaline are two sympathomimetic agents commonly used for tocolysis, although at the present time only ritodrine is approved by the FDA for this purpose. These agents, while predominantly β-2 active, do have some β-1 activity. Heart rate and cardiac output increase and myocardial ischemia or even cardiac failure can occur.

Pulmonary edema (either cardiogenic or noncardiogenic) is one of the more worrisome complications of prolonged beta mimetic therapy. Noncardiogenic causes result from changes in pulmonary capillary permeability with movement of protein into the interstitium.[7, 90] Beta mimetics stimulate the release of antidiuretic hormone (ADH) and act directly on the renal tubules to increase reabsorption of sodium.[6, 74] As a result, after 24 hours these patients are at risk for fluid overload, enhanced when intravenous saline is administered as opposed to intravenous dextrose solutions.[66] Dextrose solutions, however, are usually avoided because of the hyperglycemia induced by β-adrenergic agents. Half-normal saline is the preferred alternative. The onset of hyperglycemia is sudden, even in a nondiabetic woman. Patients with diabetes or those receiving steroids may require insulin. Type I diabetics usually require an insulin infusion to prevent diabetic ketoacidosis.[7]

β-Adrenergics produce hypokalemia, as intracellular transport of potassium is facilitated.[45] No correction is necessary, because serum potassium levels resolve after cessation of the tocolytic therapy.

When inducing general anesthesia, beta mimetics should be discontinued at least one half hour before induction if possible. The stimulation of laryngoscopy and intubation may generate severe tachycardia and hypertension. If the urgency of the case prohibits delay, esmolol can be a useful adjunct. Agents that cause tachycardia are best avoided. Halothane can sensitize the heart to catecholamine-induced dysrhythmias to a greater degree than other inhalational agents and should be used with caution, if at all. Hyperventilation should also be eschewed, as the resulting hypocarbia further decreases serum potassium levels.

If regional anesthesia is chosen it should be instituted cautiously. Prehydration may not be necessary and can result in pulmonary edema. Ephedrine may elicit an exaggerated response in heart rate and blood pressure. One possible approach is to increase the level of an epidural anesthetic slowly and administer small boluses of fluid as necessary.

Magnesium sulfate is another agent widely used for tocolysis. Like the beta mimetics, it has been associated with pulmonary edema.[32] The concerns regarding prehydration after magnesium therapy are similar to those for the β-adrenergics.

Considerations regarding general anesthesia and magnesium therapy center around the ability of magnesium to potentiate both depolarizing and nondepolarizing muscle relaxants. Precurarization is not used because the magnesium itself prevents fasciculations[29] and because its combination with even a defasciculating dose of a nondepolarizer may lead to premature paralysis. As a matter of safety, the dose of succinylcholine used for intubation should not be reduced, but the degree of relaxation should be monitored by a nerve stimulator and additional administration of relaxants administered accordingly.

Preeclampsia

The American College of Obstetricians and Gynecologists classifies hypertensive disorders associated with pregnancy into the following categories: (1) chronic hypertension, (2) transient hypertension of late pregnancy, (3) preeclampsia/eclampsia, (4) chronic hypertension with superimposed preeclampsia, and (5) unclassified.[44] Preeclampsia is characterized by the development of hypertension, proteinuria greater than 0.3 g/L/24 hrs, and nondependent edema after the 20th week of gestation. Only two of these three signs need be present to make the diagnosis. Hypertension itself is defined as (1) an increase in systolic blood pressure of at least 30 mm Hg over baseline, (2) an increase in diastolic blood pressure of at least 15 mm Hg over baseline, or (3) an increase in mean blood pressure of at least 20 mm Hg over baseline. Alternatively, hypertension can be diagnosed if, on at least two measurements 6 hours apart in the absence of external stress, one of the following is present: (1) a systolic blood pressure of at least 140 mm Hg, (2) a diastolic blood pressure of at least 90 mm Hg, or (3) a mean arterial blood pressure of at least 105 mm Hg.

Preeclampsia is one of the leading causes of both maternal and fetal morbidity and mortality. Maternal complications can include cerebral hemorrhage, hepatic rupture, cardiac arrest, pulmonary edema, and aspiration. Perinatal death is most often the result of large placental infarcts, marked placental growth retardation, or abruptio placentae.[59] Preeclampsia occurs in 5% to 10% of pregnancies and is seen more commonly in nulliparas, women of lower socioeconomic status, and women at the extremes of childbearing age. The disorder is often associated with conditions in which there is rapid uterine enlargement, as with multiple gestations, polyhydramnios, hydatidiform moles, and diabetes.[40]

Preeclampsia is referred to as severe if the systolic blood pressure is greater than 160 mm Hg, the diastolic blood pressure is greater than 100 mm Hg, proteinuria is greater than 5 g/L/24 hrs, oliguria is less than 500 mL/24 hrs, or the patient develops signs of pulmonary compromise, cerebral or visual disturbances, or epigastric pain. Eclampsia is the development of a grand mal seizure in a preeclamptic gravida in the absence of another cerebral condition. It occurs in 0.2% to 0.7% of pregnancies with one third developing postpartum. It is important to remember that the development of eclampsia cannot be predicted on the basis of a woman's presenting blood pressure. Convulsions may occur with mildly elevated blood pressure. Conversely, severely elevated blood pressure does not necessarily herald the onset of eclampsia.[31]

The severely preeclamptic patient should therefore not be rushed unprepared into the operating room for emergency cesarean section in an effort to prevent seizures. In the absence of signs of fetal compromise, the mother's blood pressure should first be stabilized, bringing the diastolic pressure to the range of 90 to 105 mm Hg. Hydralazine and labetalol are most commonly used for this purpose. Diazoxide has a lower margin of safety and may create adverse fetal complications.[17] Oral nifedipine has been found efficacious.[86] If fetal distress necessitates an emergent operative delivery and the blood pressure must be brought under control more quickly, intravenous infusions of trimethaphan or nitroprusside may be used. Obstetricians in the United States most often use magnesium therapy for prophylaxis against eclampsia, although the exact mechanism of action of this beneficial effect is a matter of controversy.

A lumbar epidural block provides safe, excellent analgesia for labor, improves uteroplacental perfusion, preserves renal function, and may improve the fetal condition. In addition, blockade to the T8 level promotes significantly reduced levels of circulating catecholamines, which are elevated in the preeclamptic woman.[47] Safe administration of regional anesthesia in these parturients requires the absence of significant coagulopathy, a diastolic blood pressure below 110 mm Hg, and an adequate circulating blood volume. The coagulation abnormalities most often present involve a low platelet count, abnormal platelet function, and prolonged thrombin time. Most coagulation parameters are not altered unless the disease is severe. If a low ($<100,000$ platelets/mm^3) or diminishing platelet count is present, one may wish to evaluate the bleeding time prior to the insertion of an epidural catheter.

Circulating blood volume is often low in severe preeclamptics and central venous pressure values of -1 to -4 cm H_2O are common.[48] Correction of hypovolemia must be accomplished before a regional block, particularly if that block is to be extended for cesarean section. Yet overhydration remains a danger in these women, who may develop pulmonary edema. For these reasons, central venous pressure monitoring is usually instituted in severe or moderately severe preeclamptic patients to assist in gauging the adequacy of fluid therapy. Intravenous fluids are given to raise the central venous pressure by 3 to 4 cm H_2O to prevent hypotension upon institution of the block. The compliance and degree of compromise of the right and left ventricles in preeclampsia, however, is often unequal.[22] Urinary output may be unreliable as a guide to hydration, as it can be compromised significantly by glomerular endotheliosis. Therefore, in cases of refractory oliguria or if signs of congestive heart failure are present, monitoring with a pulmonary artery catheter may be more appropriate than a central venous pressure line.

The level of the block should be raised slowly. Preeclamptic women usually have an increased systemic vascular resistance that can fall suddenly in the presence of a sympathetic blockade. In the event that a cesarean section becomes necessary and time permits, it would be safer to increase the level of an epidural block gradually than to institute a subarachnoid block because of the danger of sudden maternal hypotension. If hypotension does occur with an epidural block, ephedrine can be given cautiously in small doses (5–10 mg, IV) and hydration increased. Preeclamptic women may have exaggerated responses to exogenous or endogenous catecholamines.

If general anesthesia is to be used for a cesarean section, several points must be remembered. Because most preeclamptic parturients receive magnesium therapy, the same considerations apply for this drug that were mentioned in the section on tocolytic therapy. Laryngeal edema may diminish the size of the airway and create severe difficulty with intubation. Several sizes of endotracheal tubes (6.0 mm inside diameter to 7.00 mm inside diameter) should be available. The preeclamptic may have a marked hypertensive response to laryngoscopy and intubation. Antihypertensives such as trimethaphan or nitroprusside should be available to treat these reactions. Drugs such as ketamine should be avoided. Isoflurane is perhaps the volatile agent of choice in these patients. It lowers systemic vascular resistance and blood pressure while causing less cardiac depression than other agents.[79] Blood pressure must be monitored frequently but an arterial line may not be necessary if an automatic electronic device is available.

Fetal Distress

The term *fetal distress* is used and misused often. In common usage it may refer to any of a wide variety of situations in which the well being of the fetus is chronically or acutely threatened. The degree of urgency accompanying these different problems varies markedly.

The anesthesiologist's primary responsibility is to the mother. In an emergent situation, time is limited. Assessment of the patient must be performed as rapidly as possible. Priorities may dictate that the anesthesiologist forego such things as a complete reading of the patient's chart before induction of anesthesia. Communication between anesthesiologist and obstetrician becomes imperative so that each may understand the degree of urgency and relative risks involved. The history must concentrate on presence of allergies, prior anesthetic complications, medical illnesses, and current medication. Physical examination must include vital signs, assessment of the airway, and general physical condition. This must all be done in a professional manner, offering as much reassurance to the mother as time will permit.

The woman should be examined for adequate uterine displacement to avoid aortocaval compression. Antacids should be administered if there is time. Supplemental oxygen is administered for either vaginal or abdominal delivery. Blood pressure is taken frequently and hypotension treated promptly.

The presence of fetal distress per se is not a contraindication to regional anesthesia. If hypotension is avoided, regional anesthesia is not only safe but may be preferable to general anesthesia.[28, 54] The issue, however, again becomes one of time. In experienced hands a subarachnoid block can be placed swiftly. Rapid hydration with 1500 to 2000 mL of a nondextrose containing solution will usually be necessary to avoid hypotension. But if the anesthesiologist is not facile with this procedure or if the physical nature of the patient is such that difficulty in placing the block can be expected, the technique is inappropriate. If an umbilical cord has prolapsed, proper positioning of the patient to insert a subarachnoid block may not be achievable.

If a functioning epidural catheter is already in place, the use of 3% 2-chloroprocaine can quickly extend the level of the block for either an emergent vaginal or abdominal delivery. Once again, care must be taken to avoid hypotension.

If general anesthesia is to be used, 4 minutes for preoxygenation is not necessary. Four maximally deep inspirations of 100% oxygen suffice.[61] The dose of barbiturate used for induction can be reduced by using a barbiturate-ketamine combination.[54] Maternal F_{IO_2} should be 0.6 or more until delivery. Depending on the nature of the distress, supplemental oxygen given to the mother may increase oxygen supply to the fetus, but maternal partial pressure of oxygen (Po_2) must be high for a significant elevation to occur.[55]

SUMMARY

The preanesthetic assessment of the obstetric patient must take into consideration the nature of the pregnancy, the fetal status, and the normal physiologic changes of pregnancy. Some of these changes that are of particular concern to the anesthesiologist involve aortocaval compression, alterations in respiratory function, and conditions that predispose towards regurgitation and aspiration and altered anesthetic requirements.

Communication between the obstetrician and the anesthesiologist is important in formulating an anesthetic plan. This is especially true in conditions such as a breech delivery and fetal distress.

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Preanesthetic Assessment of the Pediatric Patient

*Daran Haber, MD**

A thorough understanding of physiologic and anatomic development from fetal life through adult life is essential for the preanesthetic assessment of pediatric patients. Children are not simply small versions of adults. The physiologic and anatomic characteristics necessary for life in the intrauterine environment may persist (to an extent) for months into extrauterine life, especially in the baby born prematurely. As children progress in life, their anatomy and physiology approach that of adults. Nevertheless, specific attention must be focused upon the differences among neonates, infants, children, and adults for safe anesthetic practice.

The fetus becomes physiologically mature enough to sustain extrauterine life at approximately 26 to 28 weeks' gestation. Infants born before 38 weeks' gestation are considered premature. The neonatal period encompasses the first month of extrauterine life. Infants are defined as being between 1 and 12 months' old, and children are between the age of 1 year and puberty.

ANATOMIC AND PHYSIOLOGIC DIFFERENCES BETWEEN CHILDREN AND ADULTS

Cardiovascular System

During intrauterine life the lungs are nonfunctional and the fetus acquires its oxygen from the placenta (Fig. 1A). Only enough oxygenated blood is supplied to the lungs to allow for development and maturation of the pulmonary system (approximately 5% to 10% of the cardiac output). The remainder of blood passing through the pulmonary artery is shunted through the ductus arteriosus to the descending aorta. Most oxygenated blood is transported from the placenta by the

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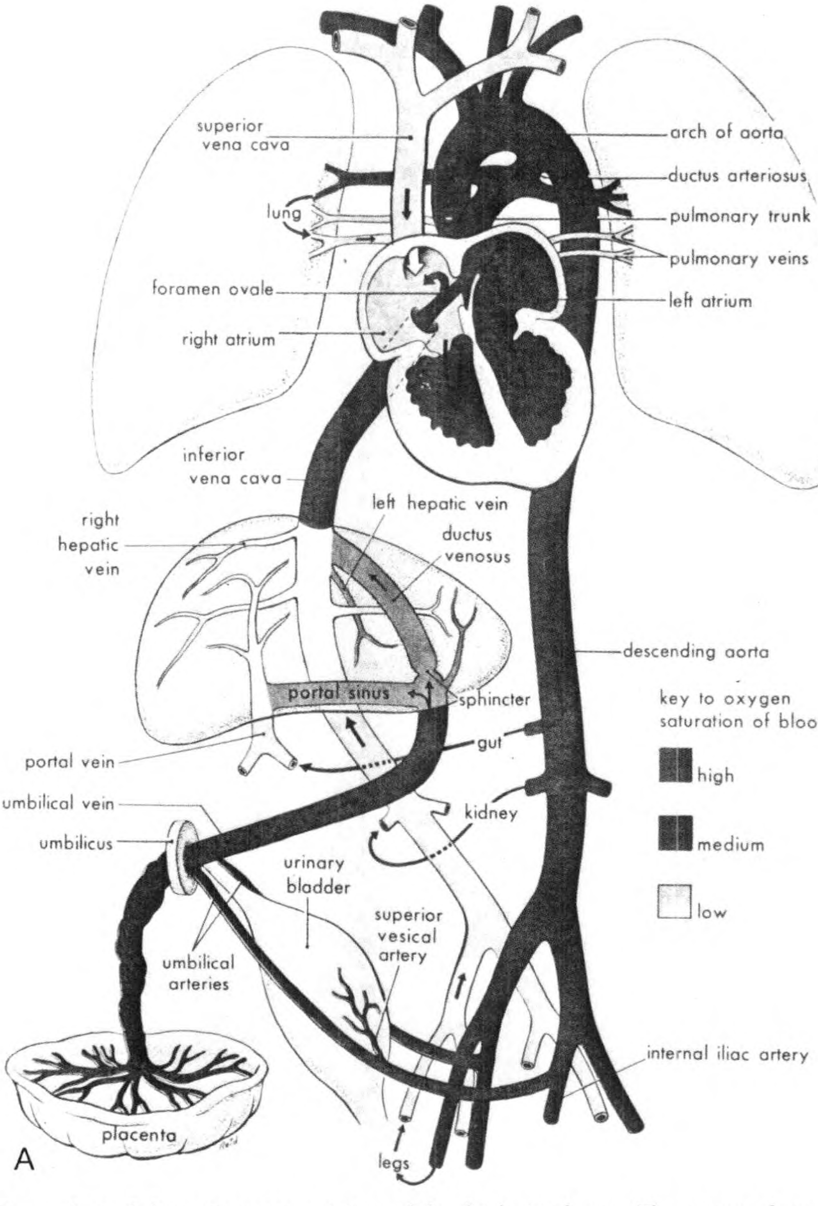

Figure 1. *A*, Schematic representation of the fetal circulation. The arrows show the course of the fetal circulation. Three shunts permit most of the blood to bypass the liver and the lungs: (1) the ductus venosus, (2) the foramen ovale, and (3) the ductus arteriosus. (*From* Moore KL: The Developing Human, ed 2. Philadelphia, WB Saunders, 1977, p 280.)

(Illustration continued on opposite page)

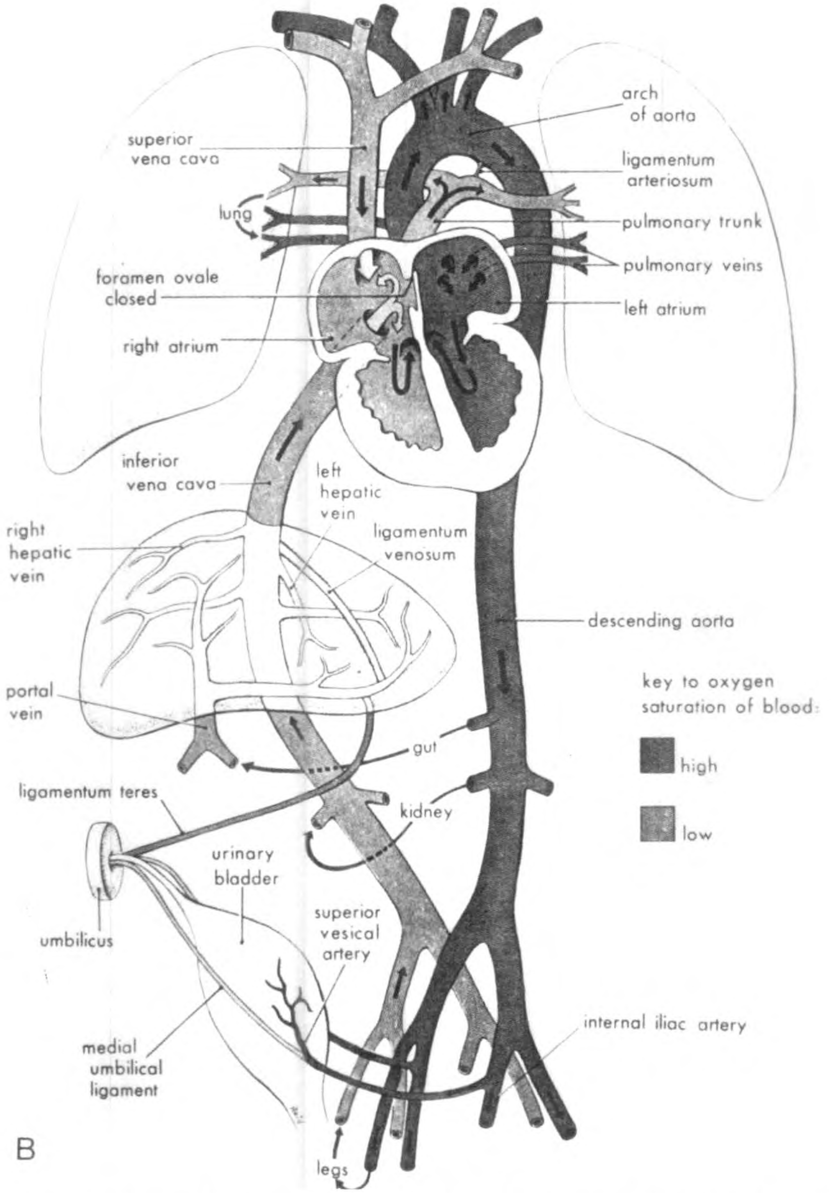

Figure 1 (*Continued*). *B*, Schematic representation of the neonatal circulation. The adult derivatives of the fetal vessels and structures that become nonfunctional at birth are shown. The arrows indicate the course of the neonatal circulation. After birth, the three shunts that short circuited the blood during fetal life cease to function, and the pulmonary and systemic circulations separate. (*From* Moore KL: The Developing Human, ed 2. Philadelphia, WB Saunders, 1977, p 281.)

umbilical vein and inferior vena cava to the heart. There it crosses the foramen ovale directly into the left atrium. In the fetus, therefore, a parallel circulation exists. At birth, as the lungs fill with air, pulmonary vascular resistance suddenly drops, and pulmonary blood flow increases, increasing left atrial volume and pressure, thereby closing the foramen ovale. Systemic arterial pressure is raised above pulmonary arterial pressure and prevents right to left shunting of blood through the ductus arteriosus.[15] Within 10 to 15 hours after birth the ductus begins to constrict. Closure of the ductus is mainly dependent upon arterial oxygenation.[22] Permanent closure occurs in 2 to 3 weeks (Fig. 1*B*). In premature infants the ductus may remain patent for a prolonged period of time with left to right shunting, causing pulmonary edema and congestive heart failure.[15]

At birth the hemoglobin content of the blood is normally high, approximately 19 g/dL, comprised predominantly of fetal hemoglobin.[13] Anemia in the neonate is defined as a hemoglobin level below 13 g/dL. The percentage of fetal hemoglobin gradually declines and by 6 months is largely replaced by adult hemoglobin. Fetal hemoglobin has a greater affinity for oxygen than adult hemoglobin, resulting in a left shift of the oxyhemoglobin dissociation curve. Because the newborn has a significantly higher oxygen consumption than adults (about 6 mL/kg/min or approximately two times the adult value), the newborn must compensate with a high hemoglobin concentration and a high cardiac output. Cardiac output in neonates is approximately 30% to 50% higher per kg body weight than that of adults.[15] The ventricles lack distensibility, and sympathetic innervation is incomplete. Cardiac output therefore depends largely on heart rate.[46] The average heart rate in a healthy neonate is about 130 beats/min. In a premature baby it is about 150 beats/min. Heart rate decreases with age, reaching adult levels by about 5 years of age[13] (Table 1). If the heart rate falls

Table 1. *Circulatory Variables in Infants and Children*

AGE	HEART RATE (Beats/min)	SYSTOLIC BLOOD PRESSURE (mm Hg)	DIASTOLIC BLOOD PRESSURE (mm Hg)	CARDIAC INDEX (L/min/m^2)	OXYGEN CONSUMPTION (mL/kg/min)
Preterm	150 ± 20	50 ± 3	30 ± 3		8 ± 1.4
Term	133 ± 18	73 ± 8	50 ± 8	2.5 ± 0.6	6 ± 1.0
6 mo	120 ± 20	89 ± 29	60 ± 10	2.0 ± 0.5	5 ± 0.9
1 y	120 ± 20	96 ± 30	66 ± 25	2.5 ± 0.6	5.2 ± 0.1
2 y	105 ± 25	99 ± 25	64 ± 25	3.1 ± 0.7	6.4 ± 1.2
5 y	90 ± 10	94 ± 14	55 ± 9	3.7 ± 0.9	6.0 ± 1.1
12 y	70 ± 17	109 ± 16	58 ± 9	4.3 ± 1.1	3.3 ± 0.6
Young adult	75 ± 5	122 ± 30	75 ± 20	3.7 ± 0.3	3.4 ± 0.6

From Goudsouzian AG: Anatomy and physiology in relation to pediatric anesthesia. *In* Katz J, Steward DJ (eds): Anesthesia and Uncommon Pediatric Disease, Philadelphia, WB Saunders, 1987, p 4.

below 80 to 90 beats per minute in the newborn, peripheral perfusion decreases, and hypoxia may occur. Because of decreased sympathetic supply to the heart, but normal cholinergic supply, the neonate is particularly sensitive to vagal stimulation and to vagolytic agents.

Respiratory System

The respiratory system begins to develop during the fourth week of intrauterine life as an outpouching of the fetal pharynx. The lower face is developed from the first branchial arch, the pharynx, larynx, and tracheal cartilage derived from the lower branchial arches.[25] The "pseudoglandular" period of bronchial and bronchiolar development is followed by the "canalicular" period of enlargement and vascularization of bronchioles and alveolar ducts, a period lasting until 25 weeks' gestation. The subsequent "terminal" period is characterized by formation of alveoli, approximation of pulmonary capillaries to the alveoli, and the beginning of surfactant production and secretion.[32] It is not until this period that the fetus is capable of sustaining extrauterine life. The premature infant may have an insufficient production of surfactant, resulting in increased alveolar surface tension and predisposing to atelectasis, ventilation perfusion mismatching, hypoxia, and acidosis. Respiratory distress syndrome in the preterm infant may result.

Alveoli are poorly developed at birth, increasing in size and number until about 8 years of age. Dead space is increased in spite of the narrow airways. Additionally, airway resistance is high in accordance with Poiseulle's law (Resistance $\sim$ 1/radius^4). Closing volume, the lung volume at which alveoli begin to close off, is high because of lack of elastic tissue.[13] The ribs are poorly ossified, flexible, and horizontal in orientation, and intercostal muscles are immature, resulting in a compliant chest wall. Chest expansion is inefficient and causes the infant to rely predominantly upon diaphragmatic breathing. Any increase in abdominal volume or pressure limits ventilation severely. Newborns (especially premature) have a low percentage of type 1 slow-twitch, high oxidative muscles that are important for sustained muscular activity, such as that of breathing. Intercostal muscle matures at 2 months, and diaphragmatic muscles at 8 months.[2] The diaphragm is the primary muscle of ventilation.[2]

Neonatal oxygen requirement ($\dot{V}O_2$) is 6 to 12 mL/kg, at least twice that of adults.[29] Alveolar ventilation is higher in infants than adults and is supported primarily by an increased respiratory rate. This requires a tremendous energy expenditure on the part of the ventilatory musculature, which contributes to the high $\dot{V}O_2$. The effect is magnified by respiratory disease, hypothermia, malnutrition, or systemic illness. Mechanical ventilation significantly reduces the work of breathing for these patients. At birth, air fills the primitive alveoli during the first breaths replacing fluid derived from the lungs, tracheal glands, and amniotic fluid.[32] During passage down the birth canal, pressure from uterine and pelvic contraction causes much of this fluid to be expelled. The cesarean-born infant does not have this benefit.

Its lungs retain a greater amount of interstitial fluid, which must later be drained through vascular lymphatic channels. The infant born by cesarean section is at greater risk to develop respiratory distress syndrome than is an infant born by vaginal delivery.[48]

The upper airway of the infant is anatomically different from that of an adult. The epiglottis and larynx are higher in position, the larynx at C4 as compared with C6 in the adult. The epiglottis is omega-shaped and extends over the vocal cords at a 45-deg angle. These anatomic differences create a conduit between the nasal pharynx and larynx that allows the infant to breathe and swallow simultaneously.[39]

Until 2 to 4 months of age, infants are obligate nasal breathers. As the child grows the larynx descends, reaching the adult level of C6 at 4 to 6 years of age, a position that facilitates development of speech, but prevents simultaneous drinking and breathing.

In the adult, the narrowest part of the upper airway is at the vocal cords. In the infant and child the airway further narrows distal to the cords, becoming narrowest at the area of the cricoid cartilage.[40] Thus, an endotracheal tube that passes the cords easily may be too large at the cricoid area and cause irritation and edema. The diameter of the trachea in the full-term neonate is 4 to 5 mm.[13] Edema producing an airway constriction of 1 mm will reduce the cross-sectional area by approximately 75% (in accordance with Poiseulle's law). Air flow resistance increases approximately 16 times. Thus, an air leak at a pressure of 15 to 25 cm H_2O is advised after intubation of an infant, and uncuffed endotracheal tubes are recommended until about 10 years of age. In the full-term neonate the distance between the vocal cords and the carina is 4 to 5 cm. During flexion or extension of the head, the tip of the endotracheal tube may move by as much as 2 cm.[13]

Table 2 lists some respiratory variables in infants and children.

Renal System

During fetal development, the placenta serves as the organ of excretion of fetal waste. Urine is produced by the developing kidney and passes into the amniotic fluid. As with the pulmonary system, renal vascular resistance is high. Renal blood flow and glomerular filtration rate are low. By 34 to 36 weeks' gestation, glomerular function is mature. The glomerular filtration rate increases rapidly during the first 2 weeks of life, then more slowly, reaching adult levels by 1 year of age.[13] Tubular maturation occurs more slowly. Sodium reabsorption is also immature and the newborn is an obligatory sodium loser.[2] Dilute urine of up to 50 mOsm/L may be excreted, but urine cannot be concentrated above 700 mOsm/L. Adults may concentrate urine up to 1200 mOsm/L.[13] Because of the inability to concentrate urine, the neonate is susceptible to hyperosmolarity in the face of dehydration or an intravenous sodium load. The differences, in renal function among infants, children, and adults are highlighted by Table 3.

Table 2. *Respiratory Variables in Infants and Children*

	NEWBORN	1 Y	8 Y	ADULT
Weight (kg)	3	10	25	70
Height (cm)	48	75	130	170
Respiratory frequency (breaths/min)	40	25	18	12
Tidal volume (v_T)				
mL	21	78	180	500
mL/kg	7	8	7	7
Dead space V_D				
mL	7	21	70	150
mL/kg	2	2	2.8	2
V_D/V_T	0.3	0.3	0.3	0.3
Alveolar ventilation				
mL/min	400	1200	2200	4000
mL/min/kg	130	120	80	60
Vital capacity				
mL	120	450	1500	4000
mL/kg	40	45	60	60
Functional residual capacity				
mL	80	250	1000	2400
mL/kg	28	25	40	35
Specific compliance C_L/FRC				
mL/cm H_2O/mL	0.04		0.06	0.05
Resting alveolar ventilation				
mL/min	400	1200	2200	3000
mL/kg/min	130	120	90	45
Hematocrit g/100 mL	55	35	40	42
PaO_2 (mm Hg)	60–90	90	95	95

From Goudsouzian NG: Anatomy and physiology in relation to pediatric anesthesia. *In* Kate J, Steward DJ (eds): Anesthesia and Uncommon Pediatric Diseases. Philadelphia, WB Saunders, 1987, p 6.

Table 3. *Renal function of Infants, Children, and Adults*

AGE	GLOMERULAR FILTRATION RATE (mL/min/1.73 m^2)	UREA CLEARANCE (mL/min/1.73 m^2)	CONCENTRATING CAPACITY (mOsm/kg)	URINE EXCRETION (mL/24 hr)
Term	38.5 (16.7–60.3)	36	600–1100	15–60
6 mo	110 (94–127.5)	22–46	700–1200	250–450
12 mo	117.5 (86–207)	71.1	700–1200	500–600
3 y	127 (89.4–164.6)	75.0 (38–112)	870–1310	500–600
5 y	127 (89.4–164.6)	75.0 (38–112)	870–1310	650–1000
12 y	127 (89.4–164.6)	75.0 (38–112)	870–1310	700–1500
23 y	127	75.0	800–1400	1000–1600

From Gregory GA: Pediatric Anesthesia ed. *In* Miller RD (ed): Anesthesia. New York, Churchill Livingstone, 1986, p 1761; with permission.

Central Nervous System

During the third week of fetal life, the central nervous system begins to develop out of the embryonal neuroectoderm. The basic morphology of the brain and spinal cord are formed during the first trimester. Neuronal development carries on during the second trimester. In the third trimester, it is accompanied by development of support structures such as glia, myelin, and vascular tissue.[23] Myelination of the spinal cord begins during midfetal life and carries on throughout infancy. At birth, neuronal tissue of the brain remains largely unmyelinated. Myelination continues until adolescence.[32] The newborn's brain is relatively poorly developed. Brain growth occurs most rapidly immediately before birth and during infancy. Nociceptive pathways in the spinal cord and brain, however, may become fully myelinated during the second and third trimesters.[1] Additionally, umyelinated fibers may carry nociceptive impulses in the mature adult.[1] It therefore appears that despite the relative immaturity of its nervous system, even the premature neonate does experience pain.

Secretion of cerebrospinal fluid occurs at a rate of about 20 mL/hr.[23] At birth, both cerebral blood flow and cerebral metabolic rate of oxygen consumption are low, increasing above adult values by 6 months of age, and returning to adult levels by adolescence[23] (Table 4). The cerebral vasculature of the newborn is extremely sensitive to hyperoxia. Cerebral blood flow is reduced by 33% at a fractional inspired oxygen concentration of 1.0 (as compared to a 12%–13% reduction in adults). During childhood, hypoxia (arterial Po_2 below 50 mm Hg) causes a rapid rise in cerebral blood flow, whereas infants require a much lower drop in arterial Po_2 (below 25 mm Hg) to bring about an equivalent rise.[9] The respiratory centers of infants react paradoxically to hypoxia. The hypoxemic infant may hypoventilate further, finally becoming apneic and bradycardic.[40]

After closure of the cranial sutures the skull forms a rigid box encasing the cranial vault. At this time, the Monroe–Kellie doctrine applies (an increase in volume of any one component of the cranial vault, i.e., blood, cerebral spinal fluid, and neuronal tissue, must be offset by a corresponding decrease in one or both of the other con-

Table 4. *The Variation of Intracranial Dynamics with Age*

	BIRTH	CHILDHOOD	ADULT
$CMRO_2$	2.3	4.5–5	3.0–3.5 mL/100 g/min
CBF	40	90–100	50–60 mL/100 g/min

$CMRO_2$ = cerebral metabolic rate of oxygen consumption, CBF = cerebral blood flow.

Data from Hollinger IB: Pediatric neuroanesthesia. Anesth Clin North Am 5:541, 1987; *and* Donnegan J: Anesthesia for pediatric neurosurgery. *In* Cottrell TE, Turndorf H (eds): Anesthesia and Neurosurgery, ed 2. St. Louis, CV Mosby, 1986, p 173.

stituents to maintain constant intracranial volume and pressure). Changes in intracranial pressure with a given change in intracranial volume demonstrate a decreased buffer capacity in an infant as compared with an adult.[23] Cerebral compliance is reduced probably because of the infant's relatively small size and small cranial vault. The anterior fontanelle, which is the largest fontanelle and the last to close, is usually obliterated by 2 years of age.[33] The frontal or metopic suture begins to fuse during infancy. Obliteration of other sutures varies widely but usually commences during the fourth and fifth decades of life.[23] Cranial sutures in the infant are not closed, and therefore initial increases in intracranial volume may be accommodated by cranial expansion with little change in intracranial pressure.[23]

In the infant and child, growth of the head parallels increase in brain size and weight and intracranial volume.

Gastrointestinal and Hepatic Systems and Metabolism

The primitive gut forms during the fourth week of intrauterine life as the roof of the yolk sac is incorporated into the embryo. The pharynx, esophagus, lower respiratory system, stomach, duodenum, liver, pancreas, and the bowel are all derived from ectoderm of the primitive gut. During the first trimester, the midgut is forced out into the umbilical cord, rotating as it returns to the abdominal cavity around the tenth week of fetal life. It is during this time that congenital defects such as omphalocele and malrotation may occur.[32]

The fetal liver becomes active at 10 to 12 weeks' of life. Its functions include glucose regulation, protein and lipid synthesis, and drug metabolism. Bilirubin and other waste products are excreted by the placenta. The neonatal liver accounts for 4% of total body weight. (In the adult, it accounts for only 2%). Nevertheless, conjugative enzyme systems are relatively immature. Hepatic metabolic activity begins to increase at about 3 months of age, peaking at 2 to 3 years of age, then slowly declining to adult values by adolescence.[13]

During fetal life, glucose is obtained from the mother by the placenta. The glucose level of the fetus is usually maintained at 70% to 80% of the maternal level.[13] Glycogen and fat stores are developed during the third trimester. At 34 weeks' gestation, 9 g of stored glycogen are present and 7% to 8% of total body weight is fat.[13] During the first several hours after birth, hepatic glycogen is depleted rapidly, leading to a drop in serum glucose level. Full-term neonates may have a glucose level of 30 mg/dL (preterm, 20 mg/dL) before they are considered significantly hypoglycemic. Nevertheless, at this level, pallor, sweating, or tachycardia may be present. At a glucose level below 20 mg/dL, apnea, convulsions, or other neurologic sequelae may ensue.[13]

After the stress of birth, catecholamine, cortisol, glucagon, thyroxine, renin, angiotensin, and aldosterone levels are high. These levels decrease during the first few weeks of life.[13, 40]

PHARMACOLOGIC DIFFERENCES BETWEEN ADULTS AND CHILDREN

Absorption, distribution, metabolism, and elimination of drugs differ in premature and full-term neonates, infants, children, and adults. Oral absorption of drugs in neonates is delayed because gastric emptying time is slow and does not reach adult rates until 6 to 8 months of age. Intramuscular absorption is variably delayed because blood flow to the peripheral musculature is slowed during the first 2 postgestational weeks.[40] Intravenous administration of drugs is unaffected by these variables. Distribution of drugs is influenced by protein binding, volume of distribution, and permeability of the blood-brain barrier as well as other factors such as hyperbilirubinemia. Infants and children have increased extracellular fluid volume. The percentage of total body water decreases with increasing age while the percentage of total body fat increases. Thus the volume of distribution of hydrophilic drugs in the young is increased and the volume of distribution of lipophilic drugs is decreased.[34] Additionally, blood supply to vessel-rich organs (such as brain, heart, liver, and kidneys) is increased. These organs make up 22% of total body volume in the neonate compared to 10% in the adult.[10] Serum protein levels are lower and protein binding is reduced, thus allowing higher free drug levels. Hyperbilirubinemia in the preterm or full-term neonate increases free drug levels by competitive binding to albumin.[40]

The neonatal blood-brain barrier is immature and central nervous system penetration of drugs is increased. Twenty percent to 100% more digoxin, phenobarbital, narcotics, aspirin, and theophylline enter the central nervous system in neonates than in adults.[15]

Both hepatic and renal metabolism and elimination are immature at birth and contribute to higher and prolonged circulating drug levels. Hepatic enzyme maturity is reached by 6 to 12 months' of age. Renal maturity is reached by 2.5 years.[48]

Uptake and distribution of inhalation agents in the neonate is influenced by the aforementioned factors as well as by an increased alveolar ventilation and decreased functional residual capacity. Minimum alveolar concentration (MAC) of volatile anesthetics steadily increases from birth until 2 to 3 months' of age then declines with increasing age (except for a slight increase during adolescence). MAC of halothane is 0.87% in neonates, 1.20% in infants, and 0.94% in adults.[28,34]

Because of the increased permeability of the neonate's blood-brain barrier and decreased metabolism, an increased sensitivity to barbiturates and narcotics may exist. Nevertheless, the induction dose per kilogram of body weight of thiopental has been reported to be the same as that for adults.[4] A recent study found the ED_{50} for thiopental to be 3.4 mg/kg for neonates and 6.3 mg/kg for infants up to 6 months' of age.[53] Nondepolarizing muscle relaxants and antagonists, while varying slightly in distribution and metabolism, may be given in equivalent doses per kilogram body weight for infants, children, and adults.[46] Neonates and infants require higher doses of succinylcholine per kilogram than do adults. To achieve muscle relaxation equivalent to that from a 1 mg/kg dose in adults, infants and children may require administration of 2 mg/kg.[46]

THE PREANESTHETIC INTERVIEW

The preanesthetic interview provides an opportunity for the anesthesiologist to evaluate the medical condition of the child or infant, to identify and address any conditions that may place the patient at increased risk for anesthesia or surgery, and to formulate an anesthetic plan. Additionally, it allows the anesthesiologist to develop a rapport with the patient and parents, discuss the anesthetic plan, and allay anxiety that may interfere with a smooth anesthetic induction and smooth postoperative recovery.

PREOPERATIVE EVALUATION

A thorough history and physical examination is an integral part of the preoperative evaluation. Areas of particular pertinence to anesthetic management such as airway, dentition, history of prior exposure to anesthetics, and possible complications on the part of the patient or family members must be elicited. Critical points in the history such as presence of cardiovascular or respiratory diseases, neurologic disorders, history of hepatic or renal insufficiency, allergies, and current medical therapy should be reviewed with the family and compared with documented records for accuracy. A thorough review of systems may identify possible sources of anesthetic complication (Table 5).[6] Records of previous anesthetic administration may disclose clinically unapparent problems unknown to the patient and family that may help to prevent otherwise unforseeable complications.

The physical examination, while no less thorough than in the case of the adult, must be approached cautiously. Children are often distrustful of strangers, and those who have already had experience with doctors may be fearful of noxious stimuli. Initially, during the examination, the child should not be taken from his parents, touched, or approached in a manner that may be deemed threatening. The initial part of the examination may simply be observation of the child during history taking or as a part of a socializing process as the child and his or her parents become acquainted with the physician. Careful attention should be focused upon the presence of any respiratory insufficiency. Wheezing, stridor, labored or erratic breathing, retractions, or nasal flaring may be obvious.[6] The skin may demonstrate cyanosis, pallor, dehydration, edema, jaundice, or signs of allergic reaction. Short neck or micrognathia or macrognathia may be an early alert to potential airway difficulties. Abdominal distention may be

Table 5. *Review of Systems—Anesthetic Implications*

SYSTEM	QUESTIONS TO ASK	POSSIBLE ANESTHETIC IMPLICATIONS
Respiratory	Bronchitis, cough, asthma	Irritable airway, bronchospasm, medication history
	Recent cold	Atelectasis, infiltrate
	Croup	Possible subglottic narrowing
	Apnea/bradycardia	Postoperative apnea/bradycardia
Cardiovascular	Murmur	Possible septal defect, avoid air bubbles in IV
	Cyanosis	Right to left shunt
	History of squatting	Possible Fallot's tetrology
	Hypertension	Possible coarctation, renal disease
	Rheumatic fever	Valvular heart disease
	Exercise intolerance	Congestive heart failure, cyanosis
Neurological	Seizures	Medications, metabolic derangement
	Head trauma	Intracranial hypertension
	Swallowing ability (chokes during feeding)	Possible aspiration, esophageal reflux, hiatus hernia
	Neuromuscular disease	Neuromuscular relaxant drug sensitivity, malignant hyperpyrexia
Gastrointestinal/hepatic	Vomiting, diarrhea	Electrolyte imbalance, dehydration, full stomach
	Malabsorption	Anemia
	Black stools	Anemia, hypovolemia
	Reflux	Treat like full stomach
	Jaundice	Drug metabolism/hypoglycemia
Genitourinary	Frequency	Urinary tract infection, diabetes, hypercalcemia
	Time of last urination	State of hydration
	Frequent urinary tract infections	Evaluate renal function
Endocrine/metabolic	Abnormal development	Possible endocrinopathy, hypothyroid, diabetes, etc.
	Hypoglycemia, steroid therapy	Hypoglycemia, adrenal insufficiency
Hematologic	Anemia	Possible need for transfusion
	Bruising, excess bleeding	Coagulopathy, thrombocytopenia, thrombocytopathy
	Sickle cell disease	Hydration, possible exchange transfusion
Allergies	Medication	Possible drug interaction
Dental	Loose teeth	Aspirate loose teeth
	Carious teeth	Possible subacute bacterial endocarditis prophylaxis

From Coté CJ, Todres ID, Ryan JF: The preoperative evaluation of pediatric patients. *In* Ryan JF, Todres ID, Coté CJ, et al (eds): A Practice of Anesthesia for Infants and Children, Philadelphia, WB Saunders, 1986, p 27.

present. Congenital anomalies may be noticeable, suggesting further inspection for other accompanying anomalies.[6]

If the child has been examined previously, much information can be obtained from the chart. All physical contact such as that involving auscultation and palpation should be explained to the child in understandable terms before it is carried out. Warming the stethoscope may make auscultation less traumatic. The examination should begin with procedures producing minimal apprehension, discomfort, or pain. Examination of the airway and discomforting procedures should be reserved for the end of the examination.

Necessary laboratory data and other objective testing depend on the nature of the surgery and the general medical condition of the patient. For minor elective procedures on otherwise healthy patients, most centers routinely recommend only a hematocrit test and urinalysis.[6] Patients with renal or hepatic dysfunction should have electrolytes measured as well as kidney and liver function tests done. Patients with a history of bleeding disorders should have activated prothrombin and partial thromboplastin times and a platelet count.

Patients receiving salicylates or other agents that inhibit platelet function may warrant obtaining a bleeding time before proceeding with certain elective surgical procedures. A chest radiograph may be appropriate for children with a recent history of pneumonia or severe respiratory infection. Children with chronic lung disease such as cystic fibrosis may warrant a room air blood gas analysis or pulmonary function tests. In children with a history of congenital heart disease, blood gas analyses as well as electrocardiogram, echocardiography, or more invasive testing may be required. Preoperative testing must be specifically indicated depending upon the patient's clinical presentation and the nature of the surgery.

THE PREMATURE INFANT

Preanesthetic assessment of premature infants must include an adequate prenatal history. Gestational age is important both by dates and by examination during prenatal care. Any maternal illnesses during or before conception and problems with delivery must be noted. Maternal drug history must be reported. Infants born to substance-abusing mothers may be prone to withdrawal. This may not manifest until 5 to 10 days after birth for infants exposed to barbiturates, benzodiazepines, or methadone. Symptoms of withdrawal include poor feeding, agitation, tremors, or seizures.[16] Maternal ingestion of high doses of acetaminophen or salicylates may produce persistent pulmonary hypertension or persistent fetal circulation during the first few days of life.[37]

Birth history is clinically relevant and must be sought. Cesarean born infants, for example, are three times more likely to demonstrate respiratory distress syndrome than are those born by vaginal delivery.[48] The birth weight should be recorded as well as Apgar scores.

Birth asphyxia may result in widespread systemic injury. Such a hypoxic insult to the heart, kidneys, central nervous system, or intestinal mucosa may place the infant at risk for an additional later complication. History of prematurity should be noted in an older infant or child. If the child had required prolonged endotracheal intubation and ventilatory support, subglottic stenosis is likely to be present as well as the possibility of persistent pulmonary disease.

During assessment of the premature infant, attention must be focused upon the ongoing development of the child's organ systems and their relative inability to adapt to certain extrauterine environmental demands. Premature infants may be at high risk for episodes of apnea and bradycardia.[44, 52] This is believed to be caused partially by lack of development and incomplete myelination in the central nervous system. Metabolic factors may also contribute to apnea, such as hypothermia, hypoglycemia, hypoxemia, hypocalcemia, and acidosis.[6] During the postoperative period any residual anesthetic agents, whether inhalational or intravenous, may be contributing factors to apnea. For this reason, narcotics are often avoided in the anesthesia of premature infants who are not expected to require postoperative ventilatory support. Intensive postoperative monitoring of these patients for a minimum of 24 hours is now routinely recommended.

Retinopathy of prematurity is a disease common to premature infants. It is characterized by retinal vascular obliteration that may be followed by increased vascularity, hemorrhage, and finally, retinal detachment. It may be exacerbated by prolonged oxygen therapy. Retinal maturation is generally complete by 42 to 44 weeks' gestation.[36] In any infant younger than the age of 44 weeks', oxygen delivery should be maintained no higher than that required for adequate tissue oxygenation. It may be prudent to delay elective surgery until the infant reaches 44 weeks' gestational age.[38]

Premature infants born with hyaline membrane disease because of decreased surfactant production may require ventilatory support involving high inspiratory pressures, positive end-expiratory pressure, and high inspired oxygen concentrations. These infants frequently develop bronchopulmonary dysplasia and chronically suffer from hypoxemia, hypercapnia, and increased pulmonary vascular resistance.[6] Positive end-expiratory pressure and high dose diuretic therapy may be required to decrease pulmonary edema and maintain adequate oxygenation.[16] Persistent pulmonary hypertension, formerly referred to as persistent fetal circulation, is a disorder characterized by persistently high pulmonary vascular resistance with right to left shunting through a patent foramen ovale and ductus arteriosus. Intrauterine aspiration of meconium caused by chronic fetal hypoxia may be associated. Additionally, persistent fetal circulation may be associated with neonatal respiratory distress syndrome or congenital diaphragmatic hernia.[2]

Premature infants are prone to reopening of the ductus arteriosus often on the third to fifth day after birth, as the infant begins to recover from respiratory distress syndrome with a consequent decrease in pulmonary vascular resistance.[16] Left to right shunting occurs, causing congestive heart failure. An audible murmur characteristically may be heard at the upper left sternal margin. It is often continuous, loudest during exhalation or apnea, and may increase in intensity during hyperventilation.[16] Treatment includes fluid restriction and diuretic and indomethacin administration. Long-standing ductus arteriosus may be associated with increased incidence of necrotizing enterocolitis. Indomethacin treatment may additionally play a role in the cause of necrotizing enterocolitis and may be a causative factor in the occurrence of spontaneous intestinal perforations. Symptoms of necrotizing enterocolitis include abdominal distension, vomiting, bloody stools, and shock.[16] Intestinal perforation is not uncommon and surgical repair is indicated.

Because of immaturity of the liver and decreased conjugating ability, the premature infant may have a higher bilirubin concentration than a full-term infant. Additionally, hypoproteinemia of prematurity and a poorly developed blood-brain barrier may increase susceptibility to kernicterus (bilirubin encephalopathy). Bilirubin concentrations as low as 10 to 15 mg/dL may cause kernicterus in an acidotic infant.[16] Peak bilirubin levels occur on the fifth or sixth days' of life in the case of premature infants, and on the second to fourth days' of life for term infants.[24] Exchange transfusion is necessary at levels high enough to cause kernicterus. Other causes of jaundice in infants include intravascular hemolysis secondary to Rh factor and ABO maternal-fetal incompatibility, hepatic dysfunction secondary to hepatitis, or glucuronyl transferase deficiency (e.g., Crigler-Najjar syndrome), biliary atresia, and chemical hemolysis secondary to vitamin K_3 overdose.[24]

THE CHILD WITH CONGENITAL HEART DISEASE

Common congenital heart defects include atrial septal defects, ventricular septal defects, coarctation of the aorta, mitral, tricuspid, pulmonic, or aortic valvular lesions, patent ductus arteriosus, transposition of great vessels, endocardial cushion defects, and tetralogy of Fallot-type lesions.[22] The lesions may be characterized by left to right or right to left shunting, obstruction or regurgitation of flow, or by some combination of these disorders. Left to right shunting leads to left and right ventricular overload and failure, pulmonary hypertension, and edema. Right to left shunting causes cyanosis. Obstructive lesions may be accompanied by primary myocardial disease such as in idiopathic hypertrophic subaortic stenosis (IHSS).[20]

In evaluation of the child with cardiac disease, signs and symptoms of congestive heart failure or cyanosis must be sought. Dyspnea or unusual fatigability, syncope, feeding intolerance, failure to thrive, or history of squatting may all present in children with poorly compensated cardiac disease. Signs of respiratory distress should be noted and followed up with appropriate laboratory tests such as arterial blood gas analyses and chest radiographs. An enlarged liver and spleen may be indicative of right ventricular failure. Level of general activity and percentile of height and weight may give an overall view of developmental retardation in the child with chronic heart disease. Cardiac medications, both those presently administered and those discontinued, should be noted.

The presence of noncardiac anomalies or diseases may suggest accompanying congenital cardiac anomalies. In these cases, an echocardiogram may be an appropriate screening test to rule out possible sources of later complications.[19] In children with Down syndrome, clinically significant congenital heart defects may be present in 12% of patients and an additional 48% may have undetected cardiac defects.[14] Common lesions in these children include common atrioventricular canal, ventricular septal defects, patent ductus arteriosus, and tetralogy of Fallot.[20] Children with Marfan syndrome have cardiac disease secondary to degenerative changes of abnormal cardiac tissue. These changes develop progressively after birth and usually manifest during the second decade of life. Lesions most commonly involve the mitral and aortic valves.[20]

Children who have had surgical correction of cardiac lesions should not be considered completely healthy. Residual effects of myocardial hypertrophy or dilatation or pulmonary hypertension may remain. Scarring and fibrosis secondary to ventriculotomy may be present. Ventricular dysrhythmias and conduction disturbances commonly occur after surgical repair of tetralogy of Fallot, ventricular septal defect, and common atrioventricular canal. Supraventricular dysrhythmias may occur after repair of transposition of great vessels and atrial septal defect. Surgical stress, ischemia, or anesthetic agents may evoke otherwise unseen dysrhythmias.[20]

Several excellent reviews[3, 19–22] present a more complete discussion of the anesthetic management of children with congenital heart disease.

THE CHILD WITH RESPIRATORY SYSTEM DISEASE

Healthy children with runny noses presenting for elective surgery may present the dilemma to the anesthesiologist of deciding whether to proceed with general anesthesia. In making this decision it is necessary to differentiate between allergic and infectious rhinitis. Recent upper respiratory infection (URI) may be associated with increased incidence of complications such as atelectasis and cyanosis.[31] Two recent studies of children receiving general anesthesia, however, found no difference in perioperative complication rate in children with and without mild upper respiratory infections.[31a,47] These children, however, may be more prone to hypoxemia in the immediate postoperative period.[47] Oxygen administration during transport to recovery is recommended.

Upper respiratory infections may represent the prodrome of a systemic viral infection such as chicken pox, mumps, or measles.[17] It may be characteristically accompanied by recent onset of purulent nasal discharge, cough, fever, sore throat, earache, or other flulike symptoms. On examination, the pharyngeal mucosa may be inflamed and tonsils may be enlarged. Evaluation of the tympanic membrane may disclose otitis media. Allergic rhinitis, which may mimic symptoms of upper respiratory infections, probably represents no increase in incidence of anesthetic complications compared with that of healthy patients. Children with allergic rhinitis tend to have clear nasal discharge and are afebrile. The nasal and pharyngeal mucosa tends to be pale and does not appear inflamed. Allergic rhinitis tends to occur seasonally and run a longer course.

The anesthesiologist may be called to evaluate an infant or child with acute upper airway obstruction. Possible causes include croup, epiglottitis, and foreign body aspiration. Treatment of each is different, and rapid diagnosis and treatment is essential, as hypoxia and death may ensue rapidly. Epiglottitis, most commonly caused by *Hemophilus influenzae* type B, involves an acute fulminant inflammation of the entire supraglottic region, including the epiglottis, arytenoids, aryepiglottic folds, and uvula.[8] Croup involves progressive inflammation and edema of the subglottic area, often including the entire tracheobronchial tree. Croup is a viral syndrome of insidious origin.[8] Epiglotittis may result rapidly in complete obstruction. Abrupt onset of high fever and sore throat often progress rapidly to dysphagia, dysphonia, drooling, and severe respiratory distress, particularly on inspiration.[30] Classically, the child presents sitting erect with chin thrust forward, drooling profusely. Croup is characterized by a more gradual onset, beginning with upper respiratory infection and progressing to inspiratory stridor and hoarseness, classically with a barking cough. The child often presents with tachypnea, labored ventilation, and cyanosis.

Foreign bodies may lodge anywhere in the respiratory system. If lodged in the esophagus or at the cricopharyngeal area, compression of the trachea from behind may occur, causing partial obstruction. Unless the object is in the larynx or trachea, stridor is frequently absent. Often, the parent fails to relay a recent history of the child playing with a small object. Aspiration or ingestion is frequently unwitnessed and may be unapparent clinically for some time.

Chest and neck radiographs, both anteroposterior and lateral, may be helpful, but aspirated foreign bodies are frequently radiolucent. In epiglottitis, lateral neck radiographs may demonstrate a swollen epiglottis similar in size and appearance to an adult thumb. In croup, anteroposterior radiograph of the neck may demonstrate a symmetrical narrowing of the subglottic airway (referred to as a "church-steeple sign"). If a possible diagnosis of epiglottitis is being considered, however, the child should not be approached or taken from his or her mother to ensure that the child remain calm and thus help prevent complete airway closure due to panic. Time should not be taken for radiographs to confirm the diagnosis, unless the child's condition is unquestionably stable. Rather, the child should be brought to the operating room by the parent in the accompaniment of the anesthesiologist (with all necessary intubation and resuscitation equipment at hand), pediatrician, and otorhinolaryngologist (with tracheostomy equipment available). In the operating room direct visualization of the epiglottis and endotracheal intubation under inhalational anesthesia may be performed. Any attempt to obtain blood from the child for analysis may be frightening to the child and provoke complete closure of the airway and inability to ventilate.

Children with congenital abnormalities of the upper airway may present a challenge to the anesthesiologist. Choanal atresia occurs when the pharynx fails to communicate with the infant's nares. Acute respiratory distress may be evident immediately after birth if the pathologic condition is bilateral, because the newborn is an obligate nasal breather.[25] Orotracheal intubation may be required until surgical repair or tracheostomy is performed. Disorders of branchial arch development such as Pierre Robin and Treacher Collins syndromes may cause difficulty with airway management and endotracheal intubation. Mucopolysaccharidoses such as Hunter-Hurler and Morquio syndromes may be characterized by airway obstruction or restrictive pulmonary disease secondary to widespread connective tissue derangements.[24] The severity of these disorders and potential for difficulty with airway management must be addressed preoperatively by the anesthesiologist.

Children with chronic or recurrent adenotonsillitis may be prone to respiratory obstruction and may be at high risk for sleep apnea or postoperative apnea.[49] Chronic pulmonary diseases such as asthma or cystic fibrosis may have profound implications for anesthetic management. Chest radiographs, blood gas analyses, and pulmonary function tests may be warranted. Preoperative optimization of treatment is imperative to reduce the risk of anesthetic complication. Patients with severe scoliosis may have restrictive pulmonary disease and neuromuscular disease may be associated.[17] Preoperative pulmonary function tests may be helpful for children with this disease who are old enough to cooperate.

PSYCHOLOGIC PREPARATION

Children admitted to the hospital for a surgical procedure may suffer persistent psychologic effects. The hospital environment, separation from parents, painful preoperative testing procedures, and forced induction of anesthesia may all be contributing factors. The child's age is obviously an important factor in how he or she perceives this experience. Newborns and infants below the age of 6 months do not appear to become distressed by separation from parents and readily accept strangers as parental substitutes.[45] Bonding between mother and infant is important for later development, however, and should not be disrupted for prolonged periods of time.[25] Children from 6 months to 4 or 5 years old are usually distressed when separated from their mothers and tend to show regression of behavior after surgery or hospitalization, including increased separation anxiety, extreme fear of doctors or dentists, poor sleeping, nightmares, and regression of toilet training.[45] At 4 to 6 years' of age separation anxiety begins to decrease and by school age, children develop a sense of autonomy that allows them to be separated from home and family with much less anxiety. As children reach adolescence they become very self-conscious and concerned about the effects that the surgery and anesthesia will have on then. Young children tend to think concretely, and things must be explained to them in terms they can understand. For example, telling young children that they will "be put to sleep" may lead them to think they will die, because that is what happened when their pet was put to sleep. Not telling children anything or lying to them will only make them mistrustful of hospital personnel in the future. Describing procedures to children in alarming detail is unnecessary. Simply and reassuringly explaining the general concepts is sufficient. Ideally, the parents and children are given information or orientation before hospital admission. Many hospitals distribute brochures explaining the hospital visit or offer tours, instructional videos, or puppet shows. While parents must understand the risks and benefits of procedures to be done, it is important for them to be reassured that the child will be well taken care of. Anxieties on the part of the parents are easily transmitted to their children.

Sources of anxiety for children depend upon their age and emotional background, but may come from unexpected areas. Children are naturally afraid of needles but may be even more upset by rectal thermometers and medications. Older children may become distressed by disrobing and donning hospital gowns.[45]

Children with chronic diseases that require extended or multiple hospitalizations are often especially fearful. Although they may initially appear secure and comfortable, they may suddenly become anxiety stricken when separated from their parents.

PREMEDICATIONS

Goals of premedication in children are anxiolysis, sedation and facilitation of induction of anesthesia, as well as vagolytic and antisialogogic effects. The use of routine premedication in children is controversial. While a smooth transition into the perioperative period and a smooth induction of anesthesia is theoretically less psychologically traumatic, no studies to date have conclusively demonstrated that sedative premedication is beneficial in this regard. Additionally, excessive or inappropriate premedication may be associated with increased morbidity. Nevertheless, most clinical anesthesiologists do premedicate children selectively.

Premedication may be given orally, rectally, intranasally, intramuscularly, or if a vein is cannulated, intravenously. When possible, injection should be avoided because it is painful and may only increase the agitation and anxiety of the child. Older children may object to rectally administered medication. Orally or intranasally administered drugs may have a bad taste or be irritating, but generally are less traumatic than either needles or rectal administraiton. Absorption is variable from all except intravenous routes.

Anticholinergic drugs such as atropine, scopolamine, or glycopyrrolate may be given orally or intramuscularly before the patient comes to the operating room. Atropine has the most vagolytic effect, scopolamine the most sedative, and amnestic and glycopyrrolate the most antisialogogic effect. Glycopyrrolate does not cross the blood-brain barrier, so it causes no sedation or amnesia. Doses are 0.02 mg/kg intramuscularly or intravenously for atropine or scopolamine and half of that dose for glycopyrrolate.[45] An anticholinergic agent should be administered to reduce secretions if intramuscular ketamine is to be given. Otherwise, atropine may be given intravenously prior to laryngoscopy and intubation or prior to administration of succinylcholine. Intravenous lidocaine may be helpful if given before intubation. In a recent study,[50] emesis occurred less frequently in children given intravenous lidocaine than in those not.

Preoperative sedation may be given depending upon the age and emotional background of the child, general state of health, and planned duration of anesthesia.

Infants younger than 1 year of age do not require preoperative sedation. Older children may receive barbiturates, benzodiazepines, narcotics, or other hypnotic agents. For children younger than 3 years of age rectal methohexital (20–30 mg/kg) or oral chloral hydrate (30–50 mg/kg), trichloroenthanol (70 mg/kg), or pentobarbital (4–5 mg/kg) may be given.[6] The child will fall asleep usually between 5 and 20 minutes after drug administration. Respiratory depression is less with these drugs than with narcotics, but nevertheless, patients should be carefully supervised. A recent study comparing intramuscular morphine, pentobarbital, and atropine with oral meperidine, pentobarbital, and atropine found both to be equally effective premedicants.[35] Diazepam suspension (0.4 mg/kg up to a maximum of 20 mg) may be given orally 1 to 2 hours preoperatively. Some centers give midazolam rectally or intranasally.[7] Intranasal sufentanil has been used successfully as a premedicant.[18] Additionally, orally administered fentanyl is still under investigation.[11] Controversy has been raised over the use of a fentanyl "lollipop" during this era of widespread drug abuse and accessibility to illicit drugs. Orally or transmucosally administered narcotics may, however, prove to be an effective premedicant for selective cases. Intramuscular ketamine (4–5 mg/kg) may be helpful in sedating children who are in a state of anxiety induced panic and otherwise uncontrollable. Sedation should ensue within 3 to 4 minutes.

FASTING

Children, as adults, are kept fasting for a period of hours before induction of general anesthesia to allow time for gastric emptying and reduce the risk of aspiration. Infants less than the age of 6 months have poorly developed glycogen stores and should be offered clear liquids such as dextrose in water or pediatric electrolyte solutions with dextrose up to 4 hours before surgery. This time should be reduced to 2 hours for premature infants.[6] Recent studies suggest that small volumes of clear liquids given 2 to 4 hours preoperatively do not affect either gastric fluid pH or volume.[41] Generally, as with adults, full liquids and solids are withheld for a period of 8 hours. For children at increased risk of aspiration because of delayed gastric motility (pyloric stenosis, hiatal hernia, recent trauma), emergencies, or anticipated difficult airway, H_2 antagonists such as cimetidine or ranitidine or nonparticulate antacids such as sodium citrate may be indicated.

PREPARATION FOR ANESTHESIA

Choice of Technique

General or regional anesthesia may be appropriate for the pediatric population. Regional anesthesia provides improved postoperative pain relief[43] and may be used in place of general anesthesia or as a supplement, reducing total anesthetic requirement. Regional anesthesia is frequently substituted for general anesthesia for lower abdominal and extremity surgery in the preterm neonate. Theoretically, the risk of postoperative apnea should be lower with regional than with general anesthesia for these patients. Postoperative apnea in an ex-preterm infant has, however, been reported both after caudal local anesthetic and caudal morphine administration.[27, 51] Infants and older children may tolerate regional anesthetic procedures and require little or no sedation. Younger children, however, may require heavy sedation or general anesthesia before the regional anesthetic is administered. Common regional blocks include spinal, caudal or epidural, penile, ilioinguinal, iliohypogastric, and brachial plexus blocks.[5, 43]

Equipment

For infants and children with less than 15 kg body weight, nonrebreathing anesthesia delivery systems such as those based upon Ayre's T-piece system are recommended. These systems provide a minimum of dead space and resistance to breathing and permit rapid titration of inspired anesthetic gas concentrations.[5] A commonly used system at present is the Jackson Rees system that incorporates a rebreathing bag exhalation valve into the expiratory limb of the Ayre's system. This essentially functions as a Mapleson D circuit. High gas flows are necessary (2.5 to 3 times minute ventilation) to prevent rebreathing and consequent hypercapnia. These high flows may contribute to significant temperature and moisture loss unless a heated humidifier is used.[40] In the Bain circuit, the expiratory limb surrounds the inspiratory limb to help warm the inspired gases. Standard adult circle circuits with CO_2 absorbing capacity may be safely used in children heavier than 15 kg. Modified circle systems with low dead space and low resistance valves are acceptable for lighter children.[40]

Endotracheal intubation may improve airway control, reduce gastric distension, and reduce the risk of gastric content aspiration. Because of distal airway narrowing in infants and children younger than the age of 10, uncuffed endotracheal tubes should be used. Tubes should easily pass the cords and cricoid area, and a gas leak should be heard at between 15 and 25 cm H_2O airway pressure.[5] Size of endotracheal tubes may be judged by the formula 4 + 0.25 (age in years) up to the age of about 8 years old). Newborns frequently require a size 3.5 endotrachael tube and premature infants a size 2.0 to 3.0. Additionally, the outer diameter of the endotrachael tube often corresponds to the nail bed of the fifth finger or the outer naris. Smaller tubes always should be kept available. Depth of the tube should be checked carefully because of the short distance between the cords and the carina. Masks of several sizes should be available as well as pediatric laryngoscope blades. Miller, Robertshaw, or Seward blades sized 0 to 2 may be helpful for neonates, infants, and small children. MacIntosh blades are appropriate for older children. Intubating stylets should be available and are recommended routinely for all rapid sequence intubations.

Temperature Regulation

Neonates, infants, and children, just as adults, are true homeotherms. In comparison to adults, however, neonates and infants are relatively poikilothermic. Their metabolic rates and surface area to volume ratios are higher, and they cannot generate significant heat by shivering until after 3 months' of age.[40] Heat loss may be evaporative, conductive, convective, or radiant. In a cold operating room and with large areas of skin and possible intraabdominal, intrathoracic, or intracranial tissue exposed to the environment as well as exposure of the airways to cold anesthetic gases, the infant may lose body temperature rapidly. Continuous measurement of temperature by skin, rectal, or esophageal probe is mandatory. The operating room should be prewarmed before the infant is brought in. Heating blankets and humidified circuits may be necessary. Intravenous fluid and irrigating solutions should be warmed. Infrared heating lamps may be helpful during induction of anesthesia. Care must be taken to prevent burning or hyperthermia by overuse of the heating apparatus.

Induction of Anesthesia

During the preanesthetic interview, induction of anesthesia should be explained to the child and parents in terms relative to their levels of understanding. Children who are not at high risk for aspiration of gastric contents may tolerate an inhalation induction with or without prior premedication. Young children may be told that they will be given a mask "like a pilot's mask" to breathe into or a tube through which to "blow up a balloon." They may be told that they will receive some "magic air" that will make them very sleepy so that they can take a nap during the procedure. The mask and bag may be brought out to the holding area to show young children so that they can experiment with it themselves. In many institutions, the child's parent is permitted to accompany the child into the operating room until induction of anesthesia is achieved, thus permitting a smoother transition period. Halothane is less pungent and less irritating to the airways than either enflurane or isoflurane and may be a better choice for induction. Scented masks may be used or scented Chap-Stick (Miller-Morton, Richmond, VA) may be painted over the mask to make the odor more tolerable. If the child objects to a mask directly over the face, the mask may be held a few inches away and gradually brought closer. Induction may be achieved with halothane in oxygen or in 70% N_2O and oxygen. If the child is sleeping, the gases may be held close to his face until a "steal" induction is achieved. Alternatively, the anesthesia circuit may be filled with 70% N_2O and oxygen and 4% to 5% halothane. One or two deep breaths will usually induce anesthesia rapidly and the concentrations may then be lowered. The electrocardiogram and O_2 saturation should be monitored during induction of anesthesia. As soon as the child is asleep, blood pressure should be taken; if taken earlier, the stimulation may interrupt the induction. Likewise, the room should be kept quiet to minimize stimulation of the child. Once asleep, a vein may be cannulated. Older children may be offered the option of an inhalation or intravenous induction. Eutectic mixture of local anesthetics (EMLA), an oily topical anesthetic cream, applied 60 minutes before venous cannulation, may be as effective as intradermal lidocaine in preventing pain from venipuncture.[42]

Fluid and Electrolyte Balance

Judgment of state of hydration of children on arrival to the operating room may be difficult. If the child fasted for 8 hours or more there will be a fluid deficit. Chronically ill children, however, may be hypovolemic or hypervolemic. Very often these children are severely dehydrated. They may have mottled skin, poor capillary refill, parched dry mucous membranes, sunken fontanelles, hypotension, tachycardia, and severe oliguria or anuria.[40] Infants and young children have poor sympathetic compensatory mechanisms and should be hydrated preoperatively. Normal fluid maintenance for infants and children heavier than 5 kg follows the equation 4 mL/kg/hr for the first 10 kg body weight, 2 mL/kg/hr for the next 10, and 1 mL/kg/hr for the remainder. Infants of 5 kg should receive 500 mL/day, of 3 kg should receive 180 to 200 mL/day, of two kilogram 200 to 400 mL/day, and of 1 kg, 120 to 140 mL/day.[40] Half of the fasting fluid deficit should be replaced during the first hour of surgery and the remainder during the next 2 hours.[40] Infants younger than 6 months' of age should receive a maintenance intravenous fluid with glucose.

Blood Component Replacement

In infants and small children, small amounts of blood loss that may easily go unnoticed may account for a significant portion of circulating blood volume. This must be replaced, either by crystalloid (at 3 times the amount of blood loss), colloid, or blood transfusion (of equal volume). Estimated blood volume is 90 to 100 mL/kg for the premature neonate, 80 to 90 for the full-term neonate, 75 to 80 for the infant, and 70 to 75 for the child.[40] Many physicians recommend transfusion at a hematocrit level of 30%. Some transfuse when blood loss exceeds 10% of circulating blood volume. The neonate, especially when born prematurely, may require earlier transfusion and may need a hematocrit level of 40% or higher for adequate tissue perfusion. Risk of transfusion reaction or of infection, however, cannot be ignored. Despite recent advances in the ability to detect hepatitis C, this disease may still be transmitted by blood transfusion. Risk of infection with human immunodeficiency virus (HIV), while extremely low, is still present and remains a source of concern to patients and family members. All of these considerations, including the overall health of the patient, tissue oxygen demand, likelihood of further blood loss, or dilutional effect of fluid administration must be considered before a blood transfusion is given. At most centers donated blood is reduced to components and often whole blood is not available. When large volumes of blood are lost, (> 1–1.5 times circulating blood volume), packed red blood cell replacement may need to be supplemented with platelets and plasma to restore clotting factors. Frequent testing of the hematocrit level, complete blood count, and electrolyte balance may be required, with the knowledge that equilibration of administered fluid and blood may take several hours to occur. In elective procedures when excessive blood loss is expected, phlebotomy is sometimes performed 1 to 2 weeks before the surgery, so that the patient's own blood is available should it be needed. Some centers allow directed blood donation from compatible family members.

SUMMARY

During the course of development from birth to adulthood, profound anatomic physiologic and psychologic changes occur. The neonate, infant, preschool and school-aged child, and the adolescent each possess unique characteristics that must be understood and addressed for the safe practice of anesthesia.

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Preanesthetic Assessment of the Neurosurgical Patient

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The preoperative assessment of patients for neurosurgery requires specific regard for the neuropathologic process. Optimal evaluation and preparation for surgery should help to reduce perioperative morbidity and (one hopes) improve outcome. Specific aspects of various neurologic disease states and their impact on the anesthetic plan are considered.

The goals of preoperative assessment in these patients are to (1) identify the neurologic disease that may affect anesthetic management, (2) recognize and treat preexisting disease processes, (3) evaluate drug therapy and possible interaction, (4) plan for surgical techniques and patient positioning, and (5) document preexisting neurologic function in structures at risk during surgery.

INTRACRANIAL TUMORS

Supratentorial

The patient who presents with an intracranial mass immediately compels inquiry regarding intracranial pressure. The contents of the cranium constitute the volume that exerts normal intracranial pressure (5–15 mm Hg). The relationship between volume and pressure in the cranial cavity is called intracranial compliance and is represented by Figure 1. As an intracranial mass expands, venous blood and cerebral spinal fluid are initially displaced. When these compensatory mechanisms are exhausted, compliance is reduced and further volume expansions elicit progressive intracranial pressure elevation.[12]

Preanesthetic assessment must concern itself with signs and symptoms of raised intracranial pressure: headache, nausea and vomiting, blurred vision, dizziness, and altered consciousness. Papille-

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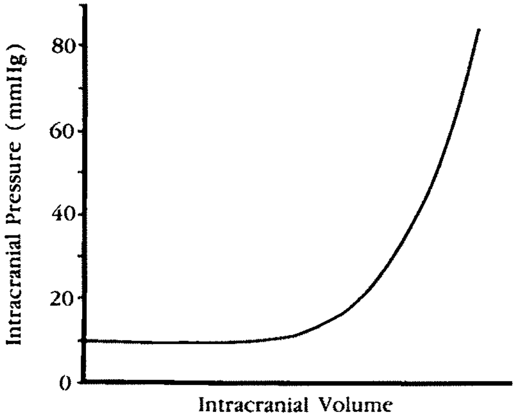

Figure 1. Idealized intracranial pressure-volume curve.

dema is diagnostic of increased intracranial pressure and indicates reduced intracranial compliance. The presence of edema and mass effect on computed tomography or magnetic resonance imaging also correlates with increased intracranial pressure.[3] As a consequence of expanding space-occupying lesions, acute elevations in intracranial pressure may produce tissue herniation and irreversible brain damage.

The standard therapy of intracranial hypertension has implications for the management of anesthesia. Fluid restriction, osmotic diuresis, and vomiting can all lead to intravascular volume depletion. Steroid treatment may predispose to hyperglycemia as well as other metabolic abnormalities.[27] Assessment of the patient's fluid status is imperative to ensure hemodynamic stability during induction and maintenance of anesthesia.

Patients with intracranial tumors may present with any form of concurrent disease. Hypertension, frequently seen concurrently, should be controlled as carefully as possible. Unrestricted hypertension in the perioperative period not only increases morbidity but may jeopardize intracranial dynamics.[22]

Pulmonary diseases must be treated preoperatively if the condition compromises the patient's ventilation and oxygenation. Hypercarbia, resulting from hypoventilation, increases cerebral blood flow and intracranial pressure. Hyperventilation is frequently used intraoperatively to decrease cerebral blood flow and intracranial pressure in combination with diuretics and cerebral spinal fluid drainage.

It is important to know the history of onset of the neurologic disease. Patients who can give this information provide insight into their present level of function. A gradual onset of symptoms relieved by steroid therapy indicates that intracranial compliance is still malleable. The sudden occurence of seizures as the presenting symptom requires that optimal therapy must be instituted before surgery. The patient who presents with a rapidly deteriorating condition has more compromised intracranial dynamics, is at greater risk for developing problems, and requires urgent therapy.

Premedication is given prudently in these patients. The most serious concern is oversedation, which may mask the development of changes in mental status. The patient who is fully alert but extremely anxious may benefit from the administration of a benzodiazepine such as diazepam, 5 to 10 mg orally, or midazolam, 2.5 to 5 mg intramuscularly. Narcotics are not recommended because of their ability to produce respiratory depression and hypercarbia. Patients who are obtunded or demonstrate changes in consciousness should not receive sedative premedication of any type.

Antihypertensive medication should be continued preoperatively, particularly beta blockers and clonidine. Anticonvulsant medication should be administered and theraputic levels documented if available. Phenytoin is selected as a first choice anesthetic because of its lack of central nervous system depression and its intravenous route of administraiton, allowing intraoperative use, if necessary. Phenytoin, 3 to 5 mg/kg, is administered to reach plasma concentrations of 10 to 20 μg/mL. Therapeutic plasma levels of anticonvulsant medication can be expected to reduce the risk of seizure in the immediate postoperative period.[18]

Patients suspected of having low-grade tumors usually receive dexamethasone, 16 mg per day, whereas patients with high-grade tumors receive 40 mg daily. An equivalent amount of methylprednisolone may be substituted for dexamethasone. Steroid administration leads to an increase in intravascular volume, hypertension, and hyperglycemia.[23]

A preoperative dose of antacid is advisable for the patient with peptic ulcer disease or one who is at risk for gastrointestinal bleeding. The obese patient may benefit from a dose of metoclopramide to decrease the volume of gastric secretions.

There is a direct association between cerebral malignancies and thromboembolic complications.[9] In a retrospective study, embolic complications occurred frequently in young, fully ambulatory, nonparetic patients. The production of procoagulants by brain tumors has been demonstrated. Some tumors appear to contain substances capable of inhibiting the fibrinolytic enzyme system. Thrombocytopenia and disseminated intravascular coagulopathy also have been identified in patients with malignancies. Thus, careful hemostatic evaluation is essential in all patients with brain lesions.

The anesthetic plan must take into consideration the patient's preoperative condition and the effects of anesthetic agents on intracranial dynamics. The agent(s) selected should decrease or have little effect on increased intracranial pressure. Systemic homeostasis and physiologic function must be maintained; wide swings in blood pressure are poorly tolerated, as patients commonly have pathologic brain regions with loss of autoregulation.[22]

A guide to the relative changes in cerebral blood flow with various anesthetic agents is shown in Table 1. A smooth induction is essential

Table 1. *All Anesthetic Agents Affect Cerebral Blood Flow and Metabolism*

DRUG	CBF	ICP	MAP	CPP
Thiopental	↓↓	↓↓	↓	↑
Fentanyl	↓	↓	SL↓	↑
Diazepam	↓	↓?	SL↓	↑
Droperidol	↓	↓	↓	SL↓
Ketamine	↑↑↑	↑↑↑	↑	↓
Halothane	↑↑	↑	↓	↓
Enflurane	↑	↑	↓	↓
Isoflurane	↑	↑	↓	↓
N_2O	↑	↑	NC	↓

Abbreviations: CBF = cerebral blood flow, ICP = intracranial pressure, MAP = mean arterial pressure, CPP = cerebral perfusion pressure.

From Newfield P, Cottrell JE (eds): Handbook of Neural Anesthesia: Clinical and Physiologic Essentials. Boston, Little, Brown, 1983; with permission.

and can be achieved with intravenous agents (with the exception of ketamine, which increases cerebral blood flow).[6] Hyperventilation is established by mask as soon as consciousness is lost. Nondepolarizing muscle relaxants are preferred because of the controversial effect of succinylcholine on intracranial pressure.[13] Chronic therapy with anticonvulsants affects the clinical duration of muscle relaxants (e.g., decreased effect of vecuronium and pancuronium).

Isoflurane may be administered before or after a smooth intubation. Of the volatile agents, isoflurane has the least effect on increasing cerebral blood flow. It depresses cerebral metabolic rate of oxygen ($CMRO_2$) more than other agents.[12] Narcotics, particularly fentanyl or sufentanil, also may be used to achieve hemodynamic stability without increasing cerebral blood flow or intracranial pressure.

The use of sufentanil is controversial at present.[11]

Infratentorial

The patient scheduled for a posterior fossa procedure should be evaluated preoperatively for signs of hydrocephalus and cranial nerve dysfunction. The signs and symptoms of increased intracranial pressure are similar, as in patients with supratentorial tumors. Because of the location of the major respiratory and cardiovascular centers in the pons and medulla, increased intracranial pressure may compress the brainstem and cause a decrease in consciousness, bradycardia, and respiratory rate.

Preoperative considerations are similar to those as described for supratentorial procedures. The choice of surgical positioning dictates many of the issues surrounding patient preparation and anesthetic technique. Controversy remains among neurosurgeons as to the best position of the patient for infratentorial surgery. The benefits of the sitting position are improved exposure because of venous and cerebral spinal fluid drainage from the field and better anatomic visualization.

Disadvantages are mainly related to adverse hemodynamic effects and the incidence of venous air embolism. The number of cases performed in the sitting position is a factor based on surgical training and anesthetic experience. Black et al[4] reported that no increased morbidity occurred in patients in the sitting or horizontal position. The patient's medical condition should certainly be weighed in this decision. Severe coronary artery disease or ischemic cerebrovascular disease is a relative contraindication to the sitting position, as marked hypotension occurs in 2% to 5% of patients.

Preparation for procedures performed in the sitting position entails the placement of a central venous catheter for monitoring and aspiration of air, which confirms the suspicion of venous air embolism. Careful patient positioning is essential to avoid nerve injury. Extremes of neck flexion must be avoided to prevent compromise of venous drainage as well as edema of the tongue and facial structures. Quadriplegia has been reported after surgery in the seated position. This has been attributed to mechanical compression of the cervical cord, reductions in blood pressure, and stretching of spinal cord vessels with neck flexion.

Anesthetic technique for surgery in the posterior fossa includes the usual concerns of maintaining cerebral perfusion and generous brain relaxation as well as a special imperative to provide a stable cardiovascular baseline. Changes in heart rhythm or blood pressure properly indicate surgical manipulation.

The prone and lateral recumbent positions are not without their particular hazards. Generally, cardiac output remains stable in the lateral position; however, venous air embolism can occur if the head is positioned above the heart. Usually the difficulties with the prone position are related to an increase in venous pressure because of obesity or poor abdominal excursion.[25]

VASCULAR DISEASE

Cerebral Aneurysms

Patients who present for surgical repair of cerebral aneurysms require skillful management throughout all phases of their hospital stay. Cerebral aneurysms occur with an incidence of approximately 15 to 20 per 100,000 people. While an occasional unruptured aneurysm may be discovered, the majority present as a subarachnoid hemorrhage. The immediate mortality rate after rupture of an aneurysm is 43%. The mortality rate increases with recurrent hemorrhage: 64% after the first rebleed and 96% after the second. Only about one third of the patients with a subarachnoid hemorrhage will be functional survivors.[19]

Controversy exists about the timing of aneurysm surgery. In an effort to prevent rebleeding, some surgeons advocate early operative intervention. Early surgery may also decrase the incidence of delayed vasospasm. If vasospasm presents quickly after rupture, with the de-

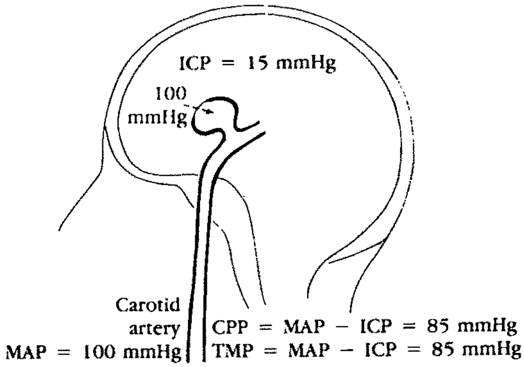

Figure 2. The transmural pressure (TMP) of the aneurysm. TMP is the same as the cerebral perfusion pressure (CPP) and is equal to the difference between mean arterial pressure (MAP) and intracranial pressure (ICP). (*From* Newfield P, Cottrell JE, (eds): Handbook of Neuroanesthesia: Clinical and Physiologic Essentials. Boston, Little, Brown, 1983; with permission.)

velopment of neurologic defecit, it may be prudent to delay surgery until it has subsided to some degree.

Regardless of the timing of surgery, goals for the preoperative evaluation are to decrease the potential for rerupture of the aneurysm and identify complications of subarachnoid hemorrhage such as vasospasm or elevated intracranial pressure. Plans are made for the provision of deliberate hypotension if necessary.

The transmural pressure in the aneurysm is defined as the difference between the mean arterial pressure and the intracranial pressure (Fig. 2). Preoperative therapy to reduce the risk of rebleeding includes the use of sedatives and the administration of antihypertensives to lower mean arterial pressure and reduce transmural pressure. Appropriate precautions must also be observed to prevent complications such as deep venous thrombosis, pulmonary embolism, sepsis, or development of metabolic abnormalities such as hyperglycemia or hyponatremia.

A grading scale for subarachnoid hemorrhage and the associated perioperative mortality rates is shown in Table 2. The presence of important systemic disease such as hypertension, diabetes mellitus,

Table 2. *Hunt's Classification of Patients with Subarachnoid Hemorrhage and Surgical Risk*

GRADE	CRITERIA	PERIOPERATIVE MORTALITY RATES (%)
I	Asymptomatic or minimal headache	0–5
II	Moderate to severe headache, nuchal rigidity, no neurologic deficit except cranial nerve palsy	2–10
III	Drowsiness, confusion, mild focal deficit	10–15
IV	Stupor, moderate to severe hemiparesis, early decerebrate rigidity and vegetative disturbances	60–70
V	Deep coma, decerebrate rigidity, moribund	70–100

chronic obstructive pulmonary disease, or severe vasospasm places the patient in the next higher grade.

Vasospasm, or vasoconstrictive angiopathy, complicates 30% of cases of subarachnoid hemorrhage, usually between days 4 and 14.[18] Although a decreased sensorium is usually associated with vasospasm, considerable arterial constriction may be observed in intact individuals. The cause is unknown but appears to be related to the presence of subarachnoid blood. Most treatment regimens include volume loading with colloid or crystalloid and cautious elevation of mean arterial pressure with vasopressors or inotropes. Nimodipine, the calcium channel blocker, has been used to treat vasospasm with varied success and is undergoing further clinical trials.[16] When nimodipine was administered within 96 hours of subarachnoid hemorrhage and maintained for 3 weeks, significant numbers of patients had good recovery compared to those who received a placebo.[17] Nimodipine may maintain calcium homeostasis to prevent the ischemic cascade of events and maintain neuronal integrity. It is now recommended to be given as soon as patients are admitted to the emergency room with subarachnoid hemorrhage.

Preanesthetic assessment requires special attention to cardiorespiratory status and evaluation of the patient's drug therapy. Electrocardiographic abnormalities are seen in 60% of patients with subarachnoid hemorrhage. The most common changes are inverted T waves, S-T segment elevation or depression, U waves, and prominent, prolonged Q-T intervals. Pathogenesis is believed to be secondary to neural, humoral (catecholamine), or metabolic dysfunction. Dysrhythmias such as sinus bradycardia or premature ventricular contractions are evident in 20% of patients.[20]

Guidelines for premedication are similar to those discussed for other neurosurgical procedures. The aneurysm patient who arrives in the operating suite with marked anxiety induced hypertension and tachycardia should receive additional sedation with benzodiazepines after oxygen and end-tidal carbon dioxide monitoring has been established.

Anesthesia should be induced slowly while continuously monitoring intra-arterial pressure. Attenuation of sympathetic responses to laryngoscopy and intubation is essential, as rupture may occur at any time before the aneurysm is clipped. Thiopental (3–4 mg/kg) or another intravenous agent of choice should allow rapid loss of consciousness. Patients may be allowed to breathe spontaneously during induction of anesthesia as hyperventilation is not required unless there is decreased intracranial compliance. Ventilation is gradually assisted as the patient receives a narcotic (fentanyl, 5–10 μg/kg, or sufentanil, 1–1.5 μg/kg) intermittently with doses of a nondepolarizing muscle relaxant. Laryngoscopy and intubation may be preceded by intravenous lidocaine, 1 mg/kg, to further attenuate any response to stimulation. To control blood pressure during induction, isoflurane may be introduced. Beta blockers such as labetolol or esmolol also may be useful for rapid treatment of hypertension or tachycardia.[21]

Maintenance of hemodynamic stability and cerebral perfusion pressure is important for optimal management.

Arteriovenous Malformations

Arteriovenous malformations are another, less common, cause of intracranial hemorrhage. The diagnosis is occasionally uncovered by the onset of seizures or severe headaches, often in younger patients. Preoperative considerations are similar to those for intracranial aneurysms. Many patients with arteriovenous malformations now undergo radiologic interventional procedures to embolize accessible vessels to decrease the size and pressure of the lesion. While this may decrease the intraoperative risk it is important to determine that renal function and intravascular volume are preserved as contrast materials are profound diuretics.

PITUITARY TUMORS

The first concern in evaluating the patient with a pituitary tumor is to ascertain whether it is functional (hypersecreting) or nonfunctional. Nonsecreting tumors are frequently larger and present problems related to their size or to a deficiency in one or more pituitary hormones.[15] Headache, visual disturbances, and increased intracranial pressure are frequent symptoms as well as hypopituitarism. The most common tumors of this category are craniopharyngiomas and chromophobe adenomas.[26]

Total pituitary deficiency in humans is probably incompatible with survival unless replacement therapy is instituted rapidly. The clinical presentation of panhypopituitarism may be dominated by either hypothyroidism or cortisol deficiency. If the syndrome occurs before puberty, short stature results.[9]

A decrease in the secretion of corticotropin (ACTH) by the anterior pituitary results in secondary adrenal insufficiency and may cause skeletal muscle weakness, weight loss, nausea, vomiting, and postural hypotension. Electrolyte abnormalities include hyponatremia and hyperkalemia.[23] Preoperative evaluation should concentrate on these problems. Anesthetic management should include the administration of exogenous corticosteroids, careful titration of anesthetic agents and muscle relaxants, and awareness of the possibility of adrenal insufficiency if hypotension occurs.

Secondary hypothyroidism is caused by anterior pituitary dysfunction and paucity of thyroid stimulating hormone. Manifestations that affect anesthetic management include a slowed metabolism of drugs, depressed baroreceptor reflexes, decreased intravascular volume, depressed ventilatory responses to hypoxia, and hypercapnia, hyponatremia, hypoglycemia, and hypothermia.[27] A posterior pituitary abnormality from tumor involvement results in a decreased secretion of antidiuretic hormone (ADH) that rarely produces diabetes insipidus. Diabetes insipidus is more common in patients with suprasellar masses that involve the hypophyseal portal system.[10] Symptoms include polyuria and polydipsia. In severe cases, hypernatremia and hypovolemia may be life-threatening. Diabetes insipidus is more commonly a postoperative occurrence. Anesthetic management includes monitoring of urine output, serum electrolyte concentrations, and serum osmolality. Diabetes insipidus may be treated intraoperatively with vasopressin tannate in oil given intramuscularly or with aqueous ADH.[15]

Hypersecreting tumors are often smaller because symptoms are produced earlier. Excess prolactin leading to galactorrhea is the most common hormonal abnormality of a hypersecreting pituitary tumor. Galactorrhea is found in 50% of cases, and the remaining patients seek medical attention because of headache.[26] No specific preoperative evaluation is required for the patient with a small prolactinoma unless there is a coexisting medical problem.

Acromegaly is caused by excess secretion of growth hormone, usually from a microadenoma of the anterior pituitary. This produces a general overgrowth of skeletal, connective, and soft tissues. Of primary anesthetic concern is the airway, as these patients may have a protruding jaw and large lips and tongue that can make laryngoscopy difficult. Cardiomegaly is frequently seen with occasional symptoms of congestive heart failure. Glucose intolerence may aggravate cardiovascular problems and decrease the lifespan.

Patients with Cushing's disease develop bilateral adrenal hyperplasia secondary to secretion of ACTH by a pituitary adenoma. They should be differentiated from patients with Cushing's syndrome, which results from tumors of the adrenal gland or the ectopic production of ACTH by nonpituitary tumors.[15] Signs and symptoms include hypertension, hypokalemia, hyperglycemia, increased intravascular fluid volume, and skeletal muscle weakness (Table 3).

Table 3. *Clinical Features of Hypersecreting Pituitary Tumors*

SYNDROME	HORMONAL EXCESS	CLINICAL FEATURES
Acromegaly	GH	Enlarged hands and feet, distortion of facial features, gigantism (prepubertal), visceromegaly, osteoporosis, glucoseintolerance, peripheral neuropathy, skeletal muscle weakness
Cushing's disease	ACTH	Centripetal obesity, "moon" facies, supraclavicular fat pad, striae, ecchymoses, hirsutism, hypertension, glucose intolerance, psychiatric disturbances
Amenorrhea-galactorrhea syndrome	Prolactin	Amenorrhea or oligomenorrhea, infertility, loss of libido

Preoperative evaluation should confirm that hypertension, hyperglycemia, and electrolyte levels are under reasonable control.

HEAD TRAUMA

Head injury secondary to motor vehicle accident or assault is currently a leading cause of death in the United States. Many patients who are brought to the emergency room with multiple trauma injuries also have head injuries as well. Many of the associated problems are generic to trauma, but the resolutions may have special and immediate ramifications for the underlying neuropathologic condition.

Early treatment of head trauma is aimed at reducing the amount of secondary injury caused by hypoxia, hypercapnia, hypotension, and expanding intracranial masses. Classification of the severity of head injury is based on the Glasgow Coma Scale (Table 4). The scale was originally used as an indicator of outcome from head trauma and as a means of assessing deterioration in intracranial status or efficacy of treatment.[24]

Hypoxia and hypercapnea must be treated promptly by administering oxygen and, if necessary, intubating and ventilating the apneic patient. Hypotension in the head-injured patient suggests another source of bleeding or spinal shock. The mechanism of injury, if known, is often a clue to the likelihood of associated injuries. Maneuvers that serve to decrease cerebral blood flow and intracranial pressure are also imperative: diuretics, 30-deg head-up position (if tolerated), hyperventilation, and barbiturate administration.

Severe brain injury activates the autonomic nervous system. Hypertension and tachycardia are the most frequently observed hemo-

Table 4. *The Glasgow Coma Scale**

Eyes Open	Spontaneously	4
	To command	3
	To pain	2
	No response	1
Best motor response		
To command	Obeys	6
To pain	Localizes	5
	Flexion withdrawal	4
	Decorticate response (abnormal flexion)	3
	Decerebrate rigidity	2
	No response	1
Best verbal response	Oriented	5
	Disoriented	4
	Inappropriate words	3
	Incomprehensible sounds	2
	No response	1

* Allowance should be made if the eyes are swollen shut or the trachea is intubated.

dynamic disorders. Although hypertension may aggravate the elevated intracranial pressure, treatment with vasodilating drugs such as hydralazine, sodium nitroprusside, and nitroglycerine may increase intracranial pressure by cerebrovasodilation. Beta blockers such as propranolol or labetolol are more appropriate.

The anesthetic plan should be the one that least interferes with intracranial pressure and maintains cardiovascular stability and cerebral perfusion pressure. If not previously performed, intubation must be accomplished expeditiously and atraumatically. Intravenous lidocaine, 1.5 mg/kg, followed by thiopental and muscle relaxants can prevent intracranial hypertension and limit cardiovascular changes. As hypocarbia is established, narcotics or isoflurane may be administered.

Approximately 50% of patients will present to the emergency room under the influence of alcohol or other drugs. It is important to consider the interactions of these substances with anesthetics and to be aware of lingering effects in the postoperative period.

CAROTID ENDARTERECTOMY

Patients with cerebrovascular disease frequently have multisystem disease that will affect their responses to anesthesia. The most commonly associated medical conditions include hypertension, coronary artery disease, peripheral vascular disease, and diabetes mellitus.[14] Transient ischemic attacks may be considered not only an indicator of carotid and cerebrovascular atherosclerosis but also a marker of serious cardiac disease and sudden death.[2]

Poorly controlled hypertension before surgery has been shown to be associated with a greater incidence of postoperative hypertension and transient neurologic deficits. Preoperative management should assess the adequacy of blood pressure control to gauge the range of normal blood pressure.

Chronic hypertension shifts the cerebral autoregulatory curve to the right and patients may need higher blood pressures to maintain normal cerebral blood flow. The adverse effects of antihypertensives include decreased sympathetic tone, alteration of response to vasopressors, and increased sedation. With beta blockers, the primary concern is the possible additive cardiac depression produced by volatile anesthetics. Similarly, the cardiac depressant effects of calcium channel blockers and volatile anesthetics appear to be additive.[27]

Antihypertensive medication should, however, be continued until the day of surgery. Profound hypertension may accompany sudden withdrawal of the centrally acting α_2-agonist, clonidine. Abrupt withdrawal of beta blockers can result in myocardial ischemia.[23]

The patient with cerebrovascular atherosclerosis is prone to concomitant involvement of the coronary arteries. In patients with unstable angina at the time of carotid endarterectomy, the mortality from myocardial infarction may be as high as 17%.[2] A combined coronary artery bypass graft and carotid endarterectomy may be considered for patients with unstable angina, left main coronary disease, or diffuse multivessel coronary artery disease. A staged procedure, carotid endarterectomy followed later by coronary artery bypass graft, may be deemed appropriate for patients with chronic, stable angina or transient ischemic attacks with severe carotid stenosis.

Chronic obstructive pulmonary disease should be suspected in patients with long tobacco smoking histories. The correlation between heavy smoking and atheromatous disease has been noted by Frost et al.[7] They reported that 40% of patients presenting for cerebrorevascular revascularization had a greater than 80 pack-year smoking history. Chronic pathologic pulmonary conditions and bronchitis are common in these patients. Decreases in arterial oxygen saturation and carbon dioxide retention are demonstrated by arterial blood gas analysis. Chest radiographs and spirometry should also be included in the preoperative evaluation. A forced expiratory volume in 1 second (FEV_1) less than 1.0 L or carbon dioxide retention indicates those patients that may benefit from preoperative therapy with bronchodilators, antibiotics, smoking cessation, chest physiotherapy, and ultrasonic nebulization.

The patient presenting for carotid endarterectomy is likely to be taking a number of medications, including antihypertensives (as mentioned), antiplatelet agents, hypoglycemic agents, antiarrhythmics, steroids, and antacids, and the anesthesiologist must be aware of the risk of complications from drug interactions. Side effects can include prolonged neuromuscular blockade, delayed return to consciousness, and altered renal and cardiac function.

A variety of anesthestic techniques have been used successfully for carotid endarterectomy, including regional anesthesia. The advantage of regional anesthesia is continuous assessment of neurologic status in the awake patient. The success of this technique is largely dependent on proper patient selection and excellent rapport among the patient, the surgeon, and the anesthesiologist. Proponents of general anesthesia cite the advantages of better airway control, maintenance of optimal blood gas values, patient comfort, better surgical exposure, and possibly some protection against cerebral ischemia. No studies, to date, have documented a difference in outcome between regional or general anesthesia.

SPINAL SURGERY

As in the brain, the spinal cord has an autoregulatory mechanism maintaining a constant blood flow. It is also subject to pathologies such as tumors, arteriovenous malformations, traumatic injury, as well as congenital abnormalities and (unlike the brain) to degenerative disease compressing the nerve roots and cord. The emphasis of the preoperative evaluation is dependent on the site and indication for surgery.

Surgery of the cervical spinal cord presents the most challenging problem for the anesthesiologist. Indications for surgery are usually degenerative disease, tumors, or cord injury. Cervical spondylosis without instability or major neurologic deficit poses no unique preoperative problems. Meticulous airway management, however, is mandatory to prevent morbidity in patients with cervical instability. Awake intubation allows control of the airway without neck movement. Psychologic preparation as well as sedation and adequate topicalization will help to facilitate the procedure. Cervical laminectomies are performed in a supine, prone, or sitting position. The airway is often not easily accessible during surgery, and obstruction or disconnection may occur. Therefore, it should be well-secured before surgery.

Although emergent spinal cord surgery is performed infrequently, anesthesiologists are often called upon to care for patients with spinal cord trauma throughout their initial and subsequent hospital stays. The anesthetic care of a patient with acute spinal cord injury requires skill and planning to avoid additional neurologic damage.

Spinal shock occurs immediately after injury and results in total absence of neural activity below the level of the lesion. Lack of sympathetic tone produces venous pooling and decreased cardiac output. Lesions that disrupt the cardioaccelerator fibers from T1 to T4 will cause bradycardia and further hypotension. Lesions below C6 to C7 leave the diaphragm intact, but there is a significant decrease in ventilatory reserve because of loss of accessory muscles of respiration. Temperature regulation is also lost below the level of the lesion, thus producing hypothermia.

Major concerns include airway function and respiratory and circulatory status to maintain adequate perfusion and oxygenation of the spinal cord.[1]

With chronic spinal cord injuries, the primary problems are the risk of autonomic hyperreflexia, hyperkalemia after succinylcholine, and the presence of hypercalcemia. Preoperative assessment should determine the level of injury and the time elapsed since the injury occurred. Autonomic hyperreflexia occurs in 85% of patients with lesions above T5. Severe paroxysmal hypertension with bradycardia occurs and is related to or initiated by visceral or cutaneous stimulation. This is best avoided by providing adequate general or regional anesthesia for procedures below the level of the lesion.

Anesthetic considerations for other procedures involving the spinal cord or vertebrae are mainly related to hazards of operating with the patient in the prone position. Prevention of congestion of epidural veins at the operative site by maintaining free abdominal movement is important in this position. Air embolism has been reported in patients undergoing surgery of the lumbar spine. Accidental damage to the aorta or iliac vessels during lumbar disc surgery may cause massive but occult blood loss intraoperatively or in the immediate postoperative period. Unexplained hypotension should alert the anesthesiologist of this rare but devastating complication.[25]

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Preanesthetic Assessment of the Ambulatory Surgery Patient

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With the increase in number of surgical procedures performed on an ambulatory basis it has become essential to have a selection process that allows for optimal performance and results. To achieve this there are two components that must be mutually compatible: the appropriateness of the surgical procedure and the patient.

The precedent for surgery on an outpatient basis was set at the beginning of this century in Glasgow, Scotland, when Nichol,[15] a pediatric surgeon, successfully completed 8988 ambulatory procedures during a 10-year period. In 1916 Ralph Waters,[20] an anesthesiologist, opened the Down-Town Anesthetic Clinic in Sioux City, Iowa and added to this precedent. Little subsequent development of such clinics occurred until the 1960s when the idea of a free-standing clinic was re-established and several units were opened, including those at University of California at Los Angeles[4] and George Washington Medical Center in Washington, DC.[12]

The American College of Surgeons approved the concept of ambulatory surgical facilities in 1983. From 1981 to 1988, the number of ambulatory surgical procedures has increased by more than 120%, and inpatient surgical volume has decreased substantially. Many free-standing centers have been established side by side with hospital related facilities, and nearly 1000 Medicare participating centers in the United States have been operational since 1988.

AMBULATORY SURGICAL PROCEDURES

Approximately 40% of total surgery[6] performed in the United States is done in an ambulatory setting, and predictions are that during

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the next few years the figure will rise to 50%. Among the procedures most usually performed on an outpatient basis are myringotomy, dilation and curettage, laparoscopy (including bilateral tubal ligation), arthroscopy, tonsillectomy and/or adenoidectomy, excision of soft tissue mass or skin lesion, cystoscopy, excision of a breast mass, bunionectomy, inguinal hernia repair, and dental procedures.

As in any other situation the evolution of acceptable procedures is constantly changing. Nonetheless, certain general characteristics must be considered in the selection process.[5] These include procedures that cause minimal blood loss and physiologic changes in body homeostasis. Another factor is duration of surgery, and normally procedures lasting longer than 90 minutes are not suitable for ambulatory surgery. Likewise, procedures suitable for training purposes must be strictly limited because of the time restraint.

Each ambulatory surgery unit establishes a list of suitable procedures that considers local conditions. An important factor is the process of third party reimbursment, as these payors issue strict guidelines on the location where particular operations can be performed.

The Patient

Initially, only the healthiest patients were considered appropriate for ambulatory surgery because it was considered essential to establish a strong safety record for this new surgical modality. Thus, class I and II patients according to the American Society of Anesthesiologists' Physical Status Classifications constituted the main patient population (Table 1). Preanesthetic evaluations were often short and inadequate. Also, as patients were young and healthy, very few complications occurred.

Since the 1960s and with increasing experience and expertise, the selection process has become less conservative, and class III patients are frequently accepted for ambulatory surgery. However, because hospital stays have been considerably shortened in this era of cost containment, anesthesiologists must often deal with patients who come to the ambulatory surgical facility on the day of surgery and who are evaluated insufficiently and prepared poorly. With this widening

Table 1. *ASA Physical Status Classification*

Class I	A healthy patient (e.g., varicose veins in an otherwise healthy patient).
Class II	A patient with mild systemic disease that in no way interferes with normal activity (e.g., controlled hypertension or diabetes or chronic bronchitis).
Class III	A patient with severe systemic disease that is not incapacitating (e.g., insulin-dependent diabetes, angina, pulmonary insufficiency).
Class IV	A patient with severe systemic disease that is a constant threat to life (e.g., cardiac failure, major organ insufficiency).
Class V	A moribund patient who is not expected to survive for 24 hours with or without surgery (e.g., intracranial hemorrhage in coma).

of patient selection there has been an increase in unplanned hospital admissions to 0.6%.[22]

The anesthesiologist will often assume the role of primary care physician in the preoperative screening of patients. In this way, the assessment is more complete and last minute cancellations are significantly reduced.

General Characteristics of the Acceptable Patient

The ideal ambulatory care patient is one in good health. If a Class III patient is accepted, the systemic condition present must be in a stable, controlled state.[9]

An essential characteristic of the patient is an ability to comprehend all aspects of the ambulatory process and a desire to participate fully. Because the patient's central nervous system will be depressed by sedation or general anesthesia, it is imperative that a responsible individual accompany the patient home and be present at all times during the overnight period. Furthermore, this person must also understand the postoperative orders and take appropriate action should an emergency arise.[16]

PREOPERATIVE EVALUATION

Ideally, the anesthesiologist's preoperative assessment is done several days prior to the planned procedure. The patient should visit the facility and meet other members of the team. An interview on the day of surgery just prior to entry to the operating room is less satisfactory.[17] A recent survey of 64 healthy patients undergoing minor gynecologic surgery, however, revealed no difference in anxiety scores, postoperative pain, or recovery times between those seen 1 to 6 days before the operation and those seen for the first time on the morning of surgery.[20] The authors considered only ASA I and ASA II patients and the group size was small. Nevertheless, the initial conclusions are surprising.

An intermediate preanesthetic assessment is a telephone interview before the day of surgery and a brief physical examination on the actual day of surgery. This has been shown in at least one study to decrease the number of surgeries cancelled from 1 in 13 to 1 in 33.[1]

A British study has formulated the essential information necessary to establish fitness for induction of anesthesia.[23] The researchers posed 11 questions, the answers to which equated with a peer consensus of suitability for anesthesia: (1) Do you feel sick? (2) Have you had any serious illness? (3) Do you get more short of breath on exertion than others of your age? (4) Do you have a cough? (5) Do you wheeze? (6) Do you have any (anginal) chest pain on exertion? (7) Do you have any ankle swelling? (8) Have you taken any medications in the past 3 months? (9) Do you have any allergies? (10) Have you had an anesthetic in the past 2 months? (11) Have you or your relatives had any problems with anesthesia?

Many ambulator surgery units now use information questionnaires for patient screening purposes based on data obtained from this study.

Recently a simple lap-top computer with four buttons (Yes, No, Not Sure, and Next Question), the Health Quiz, has been introduced to improve preoperative patient evaluation.[2, 18] The questions are displayed on a liquid crystal screen. The software is written on a credit-card-sized disk and another disk records the patient's answers. Data can be transferred to a mainframe computer for record keeping. If the Health Quiz is connected to a printer, a printout may be obtained for the patient who then may feel more enthusiastic about participating in his own care. Analyses of answers can give recommendations to the physicians about preoperative testing and appropriate management.

It is often necessary to request further evaluation from other medical specialists for sicker patients. Rather than just obtaining medical clearance, the patient receives maximum benefit when a particular health problem can be addressed and management suggestions made by the consultant.

PREOPERATIVE TESTING

The history and physical examination are the most important part of the preoperative evaluation process for the ambulatory patient. In certain circumstances further testing is helpful. Most tests that are ordered do not contribute beneficially to perioperative management.[11] With the present and ongoing concern about health care budgets, the large expenditures on laboratory tests are being questioned universally. In our opinion, approximately 12 to 18 billion dollars probably can be saved annually if only appropriate tests (those dictated by the patient's history and physical examination) are performed. While minimum requirements are mandated in certain jurisdictions before the administration of general anesthesia, these is no unanimity with regard to this testing.[3]

One plan is that used at the Montefiore Medical Center/Albert Einstein College of Medicine (Table 2). Preoperative tests "required" should be based on the operative procedure, the patient's age, preexisting medical disease, and medication history. Patients with significant baseline disease (diabetes, angina) need additional studies such as electrocardiogram, electrolyte testing, and chest radiographs.[17]

PROBLEM PATIENTS

Although problem patients are a small proportion of the total population dealt with, they provide a challenge in the screening and eval-

Table 2. *Montefiore Medical Center/Albert Einstein College of Medicine Department of Anesthesiology Minimum Requirement Preoperative Testing Order*

✓*	PATIENT'S CONDITION	HCT	CBC	PLATELET COUNT	UA	PT/PTT	SMA6	SMA12	CHEST X-RAY	ECG	PREG	SPECIMEN FOR BLOOD BANK
	Neonate											
	Patient <40 yrs.		X		X							
	>40 yrs.		X		X					X	X	
	Surg. Proc. c̄		X		X							
	blood loss		X		X	X				X		X
	CV disease†		X		X		X		X			
	Pulm disease		X		X				X	X		
	Diabetes		X		X		X			X		
	Hepatic disease		X		X							
	Renal disease		X		X	X	X	X				
	HX bleeding or											
	Hx anti-coag. use		X		X	X		X	X			X
	Smoking >20											
	pack years		X		X		X		X	X		
	Diuretic use		X		X		X					
	Digoxin use		X		X		X			X		
	Steroid use		X		X		X			X		
	Malignancy		X		X		X					
	incl. leukemias		X		X		X			X		
	Other											

* A check in the left hand column will order those labs that are marked to the right of that condition. If history indicates additional tests, you may add x-marks in any row.

† The list of diseases covers only the most common conditions. Additional tests may be required for certain patients.

uation process. Some patients may be classified as inappropriate for outpatient care. For example, patients in unstable ASA physical status 3 and 4 should be medically stabilized before surgery. Overnight hospitalization and observation is advisable for patients with a history of malignant hyperpyrexia or identified susceptibility to this syndrome. Patients who have received monoamine oxidase inhibitors may develop marked hemodynamic instability associated with anesthesia. Although the recommendation has been to discontinue these agents for 2 weeks preoperatively, such an action may be unwarranted.[7] Acute substance abuse of drugs or alcohol should preclude surgery in an ambulatory setting because of the risks of untoward cardiorespiratory responses during anesthesia. Of course, the patient who is unwilling or unable to participate in an ambulatory program cannot be forced to do so.

Some other groups of patients that may present difficulties are as follows.

The Elderly

With appropriate selection these patients are suitable for care in an ambulatory setting. The chronologic age is much less important than the physiologic age and functional state.[14] Also, in this group of patients it is essential to ascertain that the responsible individual accompanying the patient is in a suitable condition both physically and mentally to assume a custodial role toward the patient.

The Very Young

Healthy physical status I and II infants who are more than 3 months of age are considered satisfactory candidates for ambulatory anesthesia. In contrast infants with a history of respiratory distress syndrome and those born prematurely and less than 46 weeks' of postconceptual age are not suitable for day surgery. If possible, the procedures are cancelled until the infant's age and maturity are more appropriate.[13]

The Obese

This condition, common in our society, is associated with several major system disorders. Excess obesity, i.e., 35% to 40% heavier than ideal weight, is related to increased mortality after inpatient surgery. A simple formula for the ideal weight is Ideal weight (kg) = Height (cm) − 100.

The rate of unplanned hospital admission for the obese patient has been shown to be increased, although its principal reason was surgical.[10]

The Diabetic Patient

The diabetic patient can range from the youngster with brittle control to the more elderly who is maintained on a stable insulin dosage. The latter may very well be an ideal subject for ambulatory care but in all cases close cooperation is needed among the anesthesiologist and other specialists to maintain the patient in optimum condition. Ideally these patients should be scheduled early in the day and frequent blood sugar estimations must be available.

The Patient for Electroconvulsive Therapy

Patients undergoing electroconvulsive therapy are often treated on an outpatient basis. Frequently the course of treatment may require 6 to 15 visits.

Several areas in assessment of these patients require special attention including mental status, concurrent medication, and other diseases.

Informed consent for electroconvulsive therapy and anesthesia must be completed and its legality verified. The physical examination must pay particular attention to cardiopulmonary function as electroconvulsive therapy by activation of the autonomic nervous system may cause significant hypertension or cardiac dysrhythmias. Symptoms of esophageal reflux, allergies, and spinal arthritic changes should be investigated. Dentition is frequently suboptimal in the psychiatric patient and chipped or missing teeth should be noted. Bridges and loose teeth should be removed prior to electroconvulsive therapy. The ability of the patient to understand and adhere to an 8-hour fasting policy before the administration of anesthesia should be assessed.

Patients scheduled for this therapy frequently receive multiple psychotropic medications that may interact with anesthetics and muscle relaxants. For example, tricyclic antidepressants block the reuptake of norepinephrine by the presynatic membrane, leading to excess of circulating catecholamines and cardiotoxic effects such as tachycardia and dysrhythmias. An exaggerated pressor response to direct acting sympathomimetic amines may also occur, making treatment of hypotension during or after electroconvulsive therapy difficult or can cause hyperthermia, extreme hypertension, and even death. Also, central and peripheral anticholinergic effects (seen also with the phenothiazines) make glycopyrrolate a preferable drying agent to atropine. Although discontinuation of these drugs 2 weeks prior to general anesthesia has been recommended, such therapy may be ill advised for the psychiatric patient.[19] However, because many patients receive electroconvulsive therapy because medical therapy has failed, discontinuation may be reasonable. Phenothiazines exert peripheral α-adrenergic blocking action and may diminish the hypertensive response to catecholamines. A hypotensive and sedative response to barbiturates is potentiated, probably by a central action, and therefore barbiturate dose should be reduced. As noted already, patients on monoamine oxidase inhibitors are prone to hypotensive episodes during anesthesia.[7] If barbiturate doses are reduced, however, adverse effects are minimized.

Lithium is commonly prescribed for psychiatric patients. The mechanism of its antipsychotic effect is poorly understood. Nevertheless, it replaces sodium during depolarization and slows this process. Thus, lithium decreases the amount of released peripheral and central neurotransmitters. It also interacts with barbiturates to prolong recovery. The duration of depolarizing and nondepolarizing muscle relaxants is prolonged. Cardiac dysrhythmias are common.

Concurrent disease states may constitute contraindications to electroconvulsive therapy. For example, in the presence of an intracranial space-occupying lesion, a recent cerebrovascular accident, an aneurysm of any large vessel, or recent myocardial damage, electroconvulsive therapy should be performed only when it is life saving (e.g., when suicidal attempts have been made). Relative contraindications to this therapy include the presence of cardiac pacemakers, thyroid disease, glaucoma, retinal detachment, severe osteoporosis, and thrombophlebitis. Medical problems that increase the risk for electroconvulsive therapy and anesthesia include hypertension, pulmonary cardiac and liver disease, malnutrition, a history of allergic reactions, neuroleptic malignant syndrome, and the ongoing pharmacologic treatment of the psychiatric condition.[19]

UTILIZATION REVIEW

With the contemporary preoccupation over economic factors in health care, the insurance providers are intensely interested in developing utilization review programs for ambulatory surgery. These have focussed on the formation of lists of surgical procedures deemed suitable for the ambulatory unit. Also, preadmission screening criteria are formulated to justify hospital admissions.

QUALITY ASSURANCE

The motivation for a quality assurance program is improvement in patient care. In the ambulatory setting this program can provide important information by evaluating the preanesthetic screening process and unplanned hospital admissions. It provides a forum for all participants in the ambulatory unit to come together in a structured setting to discuss patient care.

CONCLUSION

The ambulatory surgery unit provides a location where the anesthesiologist uses all the skills inherent to the specialty and, in addition, may assume the role of general medical practitioner in conducting an overall examination of the patient. With an increasing volume of surgery, techniques must evolve to keep pace with the change.

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Preanesthetic Assessment of the Multiple Trauma Victim

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Trauma is a major health problem in the world today. In the United States, trauma is the leading cause of death in persons younger than the age of 38 years.[36] In terms of overall cause of mortality trauma ranks third, preceded only by cardiovascular disease and cancer. Because trauma-related morbidity and mortality strikes primarily during the most productive years of life, they have a major impact upon familial and national economic development.[36, 37] The recent medical advances in cardiovascular disease and cancer has led to a decrease in their associated mortality. In contrast, trauma-related mortality is on the rise.[37]

Death after an injury may be classified as early or late. Immediate deaths occur as a result of three types of injury: (1) approximately 50% are secondary to major cerebral or high spinal cord damage, (2) massive blood loss from major vessels or pelvic or long bone fractures, and (3) hypoxia related to airway obstruction, aspiration, or pneumothorax.[35] Late deaths primarily are caused by multiorgan systems failure and sepsis.

Improved prehospital care of trauma victims and the development of trauma centers has improved the likelihood of patient survival.[38] Emergency medical and paramedical personnel can initiate basic and often advanced life support at the trauma scene. For critically injured patients these efforts should be directed toward airway maintanence, cervical spine immobilization, fluid resuscitation, and rapid transport to a trauma center.[8]

A level I trauma center is characterized by 24-hour staffing by a trauma team of anesthesiologists, surgeons, and nurses and by the immediate availability of diagnostic and therapeutic modalities.[18]

Appropriate care of the victim upon arrival at the hospital requires an organized team approach as the initial resuscitation, evaluation,

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and stabilization occur simultaneously. The anesthesiologist performs much of the preanesthetic assessment while participating in the early stabilization procedures. The extent and accuracy of this early evaluation will allow proper and timely intervention.

GENERAL ASSESSMENT

The anesthesiologist's degree of involvement in the initial (often emergency department) evaluation of the trauma victim varies from hospital to hospital. Because airway and hemodynamic difficulties often complicate the management of trauma patients, early involvement is highly recommended. Many anesthesiologists are becoming certified in Advanced Trauma Life Support (ATLS).

Assessment of airway, breathing, circulation, and neurologic status must be performed immediately upon the patient's arrival. This evaluation generally takes about 1 minute. Asking the patient to take a deep breath can provide important information, such as level of consciousness, amount of chest trauma, and degree of airway obstruction. Likewise, recording the rate and character of the pulse is crucial. If the patient is conscious, normotensive, and without complaints of neck pain or evidence of airway compromise one can assume the patient is temporarily stable.[27] A slower, more thorough evaluation may then be performed.

During recent years several scoring systems have been developed to aid paramedical and medical personnel in quantifying the severity of injuries. The use of these systems encourage organized and periodic assessment of the patient. They also provide brief, concise, and complete means of communication to trauma team members. The Injury Severity Score is a system in which anatomic methods are used to describe the severity of injury.[2] Physiologic measures of severity include circulation, respiration, abdomen, motor, speech (CRAMS), and the trauma score.[6, 14] The trauma score is the most commonly used system of numerically describing the trauma victim's condition (Table 1). A high trauma score is encouraging, whereas a low score indicates a critical condition. A revised method of computing the trauma score excludes capillary refill and respiratory effort.[7]

The type of injury can provide additional information and help guide evaluation and management. Blunt versus penetrating injury may indicate different diagnostic techniques (e.g., identification of foreign bodies versus blunt organ damage). In addition, details about the incident may provide important clues that can help diagnose a yet unsuspected injury. For example, if the trauma patient was the driver of a motor vehicle involved in an accident is important as the driver may be more likely to have a concealed pneumothorax than the passengers.

Table 1. *Trauma Score*

TRAUMA SCORE	VALUE	POINTS	SCORE
A. Respiratory rate	10–24	4	
No. of respirations in 15 sec, × 4	25–35	3	
	>35	2	
	<10	1	
	0	0	A. ______
B. Respiratory effort		1	B. ______
Markedly shallow chest movement or air exchange	Normal	0	
Retractive-use of accessory muscles or intercostal retraction	Shallow or Retractive		
C. Systolic blood pressure	>90	4	
Systolic cuff pressure, either arm, auscultate or palpate	70–90	3	
	50–70	2	
	<50	1	
No carotid pulse	0	0	C. ______
D. Capillary refill			
Normal: forehead, lip mucosa or nail bed color refill in 2 sec	Normal	2	
Delayed: 2 sec of capillary refill	Delayed	1	
None: no capillary refill	None	0	D. ______
E. Glasgow coma scale	Ttl GCS Points		
1. Eye opening	14–15	5	
Spontaneous 4	11–15	4	
To voice 3	8–10	3	
To pain 2	5–7	2	
None 1	3–4	1	E. ______
2. Verbal response			
Oriented 5			
Confused 4			
Inappropriate words 3			
Incomprehensible words 2			
None 1			
3. Motor response			
Obeys commands 6			
Purposeful movement (pain) 5			
Withdraw (pain) 4			
Flexion (pain) 3			
Extension (pain) 2			
None 1			
Total GCS Points (1 + 2 + 3) ______			TRAUMA SCORE ______ (total points A+B+C+D+E)

The trauma score provides a means of efficiently evaluating the traumatized victim.

From Champion HR, Sacco WJ, Cormazzo AJ, et al: Trauma score. Crit Care Med 9:673, © by Williams & Wilkins, 1981; with permission.

RESPIRATORY SYSTEM

Airway Evaluation and Maintenance

Airway evaluation and appropriate oxygen therapy are paramount in the trauma victim. Every patient is assumed to have a full stomach and be at high risk for vomiting and aspiration. The conscious, spontaneously breathing patient with a patent airway deserves a thorough evaluation, which may proceed more slowly and cautiously than in other trauma patients. In contrast, the obtunded or semiconscious patient requires early airway maintenance, indications for which include airway protection, provision of a means of delivering high concentrations of oxygen, and the ability to provide positive pressure ventilation.[25]

In providing airway maintenance two difficulties are identified: (1) determining the appropriate time for intubation and (2) predicting the difficult airway. Much has been written on the difficult airway/difficult intubation, most of which is from obstetric anesthesia in which, much like trauma, mask ventilation is undesirable.[20, 24, 39] The ability to predict intubation difficulties was studied in 1984 by Cormack and Lehave.[9] They proposed gradation by laryngoscopy difficulty. This work generated Mallampati's classification to predict the difficult intubation.[19] This classification focuses on the ability to visualize pharyngeal structures with the patient sitting up with an open mouth and tongue protruding maximally (Table 2). The classification has been used successfully in preanesthetic airway evaluations and can be extrapolated to the trauma victim.

All trauma patients should receive high-flow supplemental oxygen upon arrival to the admitting area. Airway evaluation continues with determination of patency and air exchange. Visual inspection of a patient's respiratory pattern may reveal use of accessory muscles, paradoxical breathing, or other abnormal patterns. Auscultation of the chest is used to determine adequacy of air entry. Ventilation should be equal bilaterally. In patients with direct chest trauma, specific attention should be directed toward the possibility of pneumothorax and/or hemothorax.

In many patients the airway can be maintained adequately by mask ventilation with or without placement of an oropharyngeal or

Table 2. *Mallampati Classification*

Class 1	Faucial pillars, soft palate, and uvula can be visualized.
Class 2	Faucial pillars and soft palate can be visualized, but uvula is masked by the base of the tongue.
Class 3	Only soft palate can be visualized.

Depending on the ability to visualize structures an indication of the difficulty of intubation may be obtained.

From Mallampati SR, Gatt SP, Gugino LD, et al: A clinical sign to tracheal intubation: A prospective study. Can Anaesth Soc J 32:429, 1985; with permission.

nasopharyngeal airway. Certain precautions should be remembered about the use of airways. An oral airway in a conscious or semiconscious patient may invoke gagging and vomiting and thus increase the risk of aspiration. Nasal airways may be better tolerated. However, they may pierce the cribriform plate in a patient with facial or basal skull injury, however, worsening the risk of cerebrospinal fluid leakage or intracranial infection.

Endotracheal intubation is the best way to secure the airway. Several factors determine the intubation route: (1) type and severity of upper airway injury, (2) urgency of airway control, (3) experience of the intubating physician, and (4) anticipated need for long-term mechanical ventilation.[40] Direct, oral laryngoscopy with oral endotracheal intubation is the preferred approach.[25] Advantages include visualization of laryngeal structures and the ability to suction foreign bodies, inspection of upper airway and laryngeal tissues for injury, and placement of a large-diameter endotracheal tube to permit fiberoptic bronchoscopy and aggressive pulmonary cleansing to lavage. Direct, oral intubation has several potential problems. Patients will often require sedation, anesthesia, or paralysis to optimize intubating conditions, and drug administration may compromise a previously compensated individual and increase the risk of aspiration.

Nasotracheal intubation, either blind or under direct visualization, is seldom indicated. Blind attempts are hazardous because of the risk of stimulating vomiting, promoting nasal trauma and bleeding, and penetrating the brain in patients with certain types of facial or skull fractures.[34]

Fiberoptic intubation may be advantageous when the need for intubation is not immediate. Skilled bronchoscopists can secure an airway with minimal patient movement or discomfort under topical anesthesia when the patient is cooperative. This technique has particular advantages in patients with cervical spine injuries.

Despite all the currently available means of securing an airway, certain patients will, of necessity, require less conventional methods. Cricothyrotomy and tracheostomy may be necessary. Recently, transtracheal jet ventilation by a cricothyroid large-bore intravenous catheter has been proposed when desperate emergencies arise.[3]

Maxillofacial Injury

Maxillofacial trauma often produces complex problems related to airway management and respiratory function. The anesthesiologist is challenged to recognize the extent of bony, cartilaginous, and soft tissue injury. The most important function of the anesthesiologist in facial trauma is to secure the airway. Nevertheless, pain and trismus may hinder thorough evaluation of the airway. In addition, mechanical compromise may be present, and thus, differentiation is critical. Although adequate anesthesia and muscle relaxation will overcome pain and trismus, an anatomically altered jaw mandates awake intubation.

Maxillofacial injuries are classified according to the Le Fort system. Le Fort I is a fracture of the maxilla involving the floor of the nose. Le Fort II is a pyramidal fracture of the midface, which may have a break in the cribriform plate. The Le Fort III is a complete separation of cranial and facial structures. It involves the ethmoid bone, and a basal skull fracture may be present.[15] Whenever basal skull integrity is violated nasal instrumentation is contraindicated.

Thoracic Trauma

Blunt chest trauma poses several diagnostic problems. Blunt injury most frequently causes rib fractures and leads to such problems as tension pneumothorax, flail chest, or massive hemothorax. Because of the closed cavity these conditions may be unrecognized until decompensation occurs. The anesthesiologist should always suspect these potential injuries.

Conversely, with penetrating injuries the primary site is often apparent. The most common finding in penetrating injuries is a combined hemopneumothorax, with isolated conditions appearing less frequently. Open pneumothorax resulting from a penetrating injury is usually obvious. The sucking chest wound equilibrates atmospheric and intrathoracic pressures and rarely causes immediate decompensation. In contrast, a closed pneumothorax produces a one-way valve through which air exits lung parenchyma and accumulates within the thoracic cavity. Consequent tension pneumothorax can cause catastrophe if not decompressed. Clinical suggestions of a tension pneumothorax include tracheal deviation, unilateral breath sounds, unilateral tympany, hypotension, and jugular venous distension. Unsuspected pneumothorax may first appear during positive pressure ventilation under general anesthesia as increased inspired pressures, tachycardia, hypotension, and decreased breath sounds.

Hemothorax may be isolated or combined with a pneumothorax. Hemothorax associated with hypotension not responding to volume resuscitation must be explored operatively.

Pulmonary contusions are common with blunt trauma and are often concomitant with multiple rib fractures. Radiologic changes appear within the first few hours. Ventilation-perfusion mismatching produces severe hypoxia, and is life threatening. Mechanical ventilation must be continued with high oxygen concentrations and positive end-expiratory pressure if necessary.[5]

Diagnosis of these conditions is often difficult. Routine chest radiograms are frequently suboptimal and unrevealing. Maintaining a high index of suspicion will alert the anesthesiologist to severe pulmonary or vascular injury. Signs of such injury include first or second rib fractures, widened mediastinum, and flail chest.[4]

CIRCULATORY STATUS

Shock

Shock can be defined as a state of decreased tissue perfusion. The general state of shock has many causes, however, hypovolemia cer-

I. Hypovolemia
  A. Overt Blood Loss
    1. major lacerations
    2. minor lacerations with large vascular tissue (e.g., facial)
  B. Concealed Blood Loss
    1. chest
    2. abdomen—intraperioteneal
    3. major orthopedic—pelvis; femor
    4. retroperitoneal
II. Traumatic Cardiac Failure
  A. Tension pneumothorax
  B. Cardiac tamponade
  C. Cardiac contusion
  D. Dysrhythmias
III. Additional Causes
  A. Myocardial infarction
  B. Spinal cord injury
  C. Hypothermia

Figure 1. Etiology of traumatic shock. The causes can be divided into three main categories.

tainly is predominant in trauma patients (Fig. 1). Clinical manifestations of shock include anxiety, pallor, cool extremities, cyanosis, tachycardia, hypotension, diaphoresis, oliguria, cardiac dysrhythmias, acidosis, and disseminated intravascular coagulopathy.[23]

Hypovolemic shock usually is the result of severe blood loss, either internal or external. External hemorrhage is best controlled by the use of direct pressure, tourniquets, pneumatic compression, or, as a last resort, vascular clamps. Concealed blood loss occupies one of four compartments (Fig. 1). Thoracic blood loss may be identified by physical examination and, if time allows, by radiographs. Diminished or absent breath sounds with dullness to percussion should alert the anesthesiologist to possible hemothorax. Thus, in the presence of hypotension, tube thoracostomy should be performed and drainage monitored closely. Confirmation of intraperitoneal hemorrhage is made by peritoneal lavage. Femoral fractures can generate a blood loss of 500 to 1500 mL and should be controlled by external traction or pneumatic splints. Blood loss from pelvic fractures is large and difficult to control. Pneumatic trousers may be helpful in providing pressure control. Diagnosis of retroperitoneal hemorrhage is suggested by hematuria, flank ecchymosis, and enlarging flank masses.

The body's physiologic mechanisms to combat hypovolemia are geared toward maintaining blood pressure and tissue perfusion. Tachycardia and vasoconstriction result from sympathetic nervous system activity, renin-angiotensin system activation, and secretion of antidiuretic hormone. Compensation for blood loss may be adequate until approximately one third of intravascular volume has been lost.

Table 3. *American College of Surgeons' Classes of Acute Hemorrhage*

	CLASS I	CLASS II	CLASS III	CLASS IV
Blood loss (mL)	≤750	1000–1250	1500–1800	2000–2500
Blood loss (%)*	≤15	20–25	30–35	40–50
Pulse rate†	72–84	>100	>120	≥140
Blood pressure (mm Hg)‡	118/82	110/80	70–90/ 50–60	<50–60 systolic
Pulse pressure (mm Hg)	36	30	20–30	10–20
Respiratory rate	14–20	20–30	30–40	>35
Urine output (mL/hr)§	30–35	25–30	5–15	negligible
CNS-mental status	slightly anxious	mildly anxious	anxious and confused	confused and lethargic
Fluid replacement (use 3:1 rule for fluid resuscitation)	crystalloid	crystalloid	crystalloid + blood	crystalloid + blood

* Percent of blood volume in a 70-kg adult.
† Assume normal of 72/min.
‡ Assume normal of 120/80.
§ Assume normal of 40–50 mL/hr.
Classification of hemorrhage dictates evaluation and therapeutic modalities.
Adapted from American College of Surgeons Committee on Trauma ATLS™ Student Manual, 1988; with permission.

Therefore, the normotensive patient with other manifestations of shock may have profound volume depletion and should be resuscitated aggressively. Recognition of this compensation is critical to the preanesthetic management, because inhibition of nervous system activity from anesthetic agents may produce severe cardiovascular decompensation.

Quantification of hemorrhagic shock helps determine management. To this end, the American College of Surgeons has developed a classification system based upon volume lost[1] (Table 3).

Class I hemorrhage is a loss of intravascular volume of approximately 15%. The young, healthy patient can often compensate for this loss, thus making diagnosis difficult as hemodynamic parameters are usually within normal limits.

Class II hemorrhage is loss of 20% to 25% of circulating blood volume. At this stage, hemodynamics begin to change, and tachycardia, tachypnea, and narrowing of pulse pressure occur. Restoration of intravascular volume should not be delayed.

Blood loss of 30% to 35% (class III hemorrhage) crosses the point of compensation in the healthy patient. More severe hemodynamic alterations occur. Hypotension, tachycardia (greater than 120 per minute), and oliguria are present. Confused sensorium resulting from inadequate cerebral perfusion in a previously oriented individual may develop. Red blood cell transfusion is required to restore circulating volume and oxygen carrying capacity.

Class IV hemorrhage represents the extreme case of acute blood loss at greater than 40%. All of the classic signs of shock are present and severe. Resuscitation requires massive volume transfusion, primarily with red blood cells.

Volume Resuscitation

Most trauma victims require volume resuscitation. Catheter location, size, and type are as important to successful resuscitation as is the fluid type.[21] Upper extremity infusions in the patient with a thoracic injury may only worsen the situation. Infusate can extravasate and collect in the thoracic cavity. Likewise, lower extremity infusions in the patient with pelvic or abdominal vascular injuries are of little benefit. The situation is simplest with isolated injuries; multiple injuries often require both upper and lower body vascular access.

Peripheral vascular access is a problem in the hypovolemic vasoconstricted patient. The Maryland Institute of Emergency Medical Services System (MIEMSS) advocates the use of a long antecubital cannula (to infuse fluids and to measure central venous pressure) in conjunction with a saphenous line if significant cardiovascular instability is present.[10] Alternatively, central venous access may be obtained by percutaneous venipuncture of the subclavian or internal jugular veins. Risks and benefits of either approach must be considered carefully.

Catheters with good flow characteristics, according to Poiseuille's law, are large bore and of relatively short length. Peripheral choices are 14- or 16-gauge intravenous catheters. Lately, emergency infusion devices, direct insertion of intravenous tubing, or size 10- or 12-feeding tubes are used for large volume infusion. Pulmonary artery catheter introducers may be placed centrally through a percutaneous approach for massive infusion.

The choice of infusate is still controversial. Some believe initial resuscitation should utilize colloids, while others believe balanced salt solutions are safe and equally effective when administered in a 3:1 ratio (3 mL of crystalloid for every 1 mL of blood loss).[10, 13, 21] There is little debate that severe hemorrhage (class III–IV) requires blood therapy. If type-specific blood is unavailable, type O, Rh negative (or even Rh positive) blood can be used temporarily. The importance of warmed infusions cannot be overemphasized. Hypothermia is a real problem for the trauma patient and all available means should be used to prevent or reverse dangerous temperature levels.

Thoracic Vascular Injury

Thoracic vascular injury is most often the result of blunt deceleration trauma (e.g., motor vehicle accidents). Aortic rupture frequently causes death at the accident scene from blood loss. The incidence of aortic rupture in victims ejected from the automobile is more than twice that of those not ejected. The most frequently seen site of injury is along the upper portion of the descending aorta just distal to the left subclavian artery. Of all the possible areas of aortic injury, patients with this injury tend to reach the hospital alive and account for 95% of surgically treated traumatic aortic rupture.[17] Diagnosis is difficult, and a high index of suspicion is necessary, as clinical signs may not be apparent. Radiographic findings suggestive of aortic rupture include an ill-defined aortic knob and a widening mediastinum. These findings mandate angiography to visualize the vasculature and confirm diagnosis. Angiography should be performed in all patients with these findings unless clinical deterioration prevents it. One should remember that traumatic aortic rupture occassionally occurs in patients who have normal chest radiograms.[10] Other vascular injuries may occur but are less common.

Traumatic disruption of the aorta requires prompt surgical intervention. Perioperative management relates to airway maintenance and volume resuscitation as necessary. Specific attention should be directed toward avoiding hypertension; this is done with the use of antihypertensive agents. The target systolic blood pressure is $\leq$ 120 mm Hg.[10] Short acting, titratable agents are most appropriate as volume status may be precarious. Direct arterial monitoring of proximal aortic pressures is obtained with a right radial arterial line.

Penetrating injuries from gunshot wounds or stab wounds frequently result in death before the victim reaches the hospital.[10] Of those who reach a medical facility, hemorrhagic shock and hemothorax are the most common findings. Early control of the airway is essential; patients should be intubated and ventilated before hematoma formation distorts airway anatomy, making subsequent intubation difficult or impossible.

Cardiac Trauma

As with other organ damage, blunt cardiac trauma causes more tissue damage than penetrating cardiac trauma.[27] Cardiac tamponade, cardiac contusion, and cardiac rupture are the most commonly seen injuries. Classic myocardial tamponade presents with hypotension, distended jugular veins, and muffled heart sounds, i.e., Beck's triad. Additional signs are tachycardia and pulsus paradoxis. Initial management consists of volume resuscitation, inotropic support, and pericardiocentesis. Bloody pericardiocentesis with subsequent clinical improvement confirms the diagnosis.

Patients with significant myocardial contusions may present with angina pectoris, dysrhythmias, or ventricular failure.[10] Chest pain is frequently resistant to nitrate therapy.[27] Management of these patients is similar to that of those with a myocardial infarction. Hemodynamically unstable patients or those with evidence of myocardial dysfunction should be monitored by a pulmonary artery catheter to optimize care.

NEUROLOGIC EVALUATION

The central nervous system (CNS), which is composed of the brain and spinal cord, is a major concern in the initial assessment of the multiple trauma patient. Either the brain or spinal cord may be damaged by blunt or penetrating trauma. Associated morbidity and functional impairment ranges from a minimal deficit to incapacitation. By the time the neurologically injured patient reaches the hospital the insult from injury has occurred and little can be done to reverse its effects. Management, therefore, is directed toward preventing secondary central nervous system damage by providing optimal physiologic support.[12] The patient with an altered level of consciousness after an injury must be considered to have raised intracranial pressure. Likewise, those patients with deceleration injuries have spinal damage until proved otherwise.[10] Central nervous system injury must be looked for because many of its signs may be subtle. A concise, thorough evaluation is mandatory immediately upon the patient's arrival.

Initial neurologic assessment should focus on the level of consciousness, examination of eye movement, pupil size and responsiveness, and brainstem function. The level of consciousness is critical in assessing the extent of injury.[12] Metabolic factors may be responsible for a depressed sensorium. Common associated metabolic factors are hypoxemia, hypothermia, and use of recreational drugs, alcohol, and sedatives. Intracranial hypertension must be excluded before these other causes of central nervous system depression can be considered. A combination of metabolic factors and intracranial injury often coexist in the trauma victim. The Glascow Coma Scale, described previously as a component of the overall Trauma Score, has been devised to assess, prognosticate, and evaluate therapy in neurologic trauma (see Table 1). The Glascow Coma Scale incorporates three modalities: verbal response, ocular movements, and motor response.[33] This scale has become a widely accepted means of judging severity of injury, overall gross location (brainstem versus cerebral), and clinical response to therapy.

The goals of neurologic stabilization are airway establishment, ventilatory support, cardiovascular stability, and maintenance of favorable intracranial dynamics. Hypoxia frequently accompanies a severe head injury and can worsen a neurologic insult.[30] Early intervention with supplemental oxygen and endotracheal intubation with controlled ventilation may be life-saving. Laryngeal manipulation can cause an enormous increase in intracranial pressure. These changes can be alleviated by the judicious use of barbiturates, intravenous lidocaine, and short acting antihypertensive agents. Endotracheal intubation is indicated for comatose patients, as they are at increased risk of aspiration.

Cardiovascular stability is reflected by adequate cerebral perfusion pressure (CPP) and is defined as mean arterial blood pressure minus central venous pressure (or preferably intracranial pressure). The normal range for CPP is 70 to 110 mm Hg. Both prolonged systemic hypotension and hypertension can worsen neurologic deficits by causing ischemia and cerebral edema, respectively. The multiple trauma patient requires adequate volume resuscitation, which may require invasive hemodynamic monitoring with a central venous pressure or pulmonary artery catheter to maintain desirable intracranial dynamics. Most patients require areterial pressure monitoring. Hypertension and tachycardia are the most frequently observed hemodynamic abnormalities after a head injury. After adequate intravascular volume has been assured, hypertension, in excess of 30% above normal mean values, should be treated with adrenergic blocking agents.

Increased intracranial pressure is associated with about 50% of all deaths from head trauma. Normal intracranial pressure is 10 to 15 mm Hg. Intracranial pressure can be monitored by four methods: venticula catheter, subdural bolt, epidural transducer, or subdural catheter. Mechanical ventilatory support with hyperventilation can improve intracranial hypertension by constricting cerebral vasculature and therefore decreasing overall cerebral blood volume. Hyperventilation should be targeted toward maintaining partial pressure of arterial carbon dioxide (Pa_{CO_2}) between 27 and 33 mm Hg. The benefits are most apparent within the first 5 minutes then intracranial pressure slowly rises, stabilizing at a lower than original level after 3 to 5 hours. Other means of altering intracranial pressure include diuretic therapy with mannitol with or without furosemide, barbiturate administration, β-adrenergic blockade, and positioning to promote venous drainage.

Figure 2 demonstrates the cycle leading to secondary central nervous system insult. Appropriate assessment and stabilization should interrupt this process.

Cervical Spine Evaluation

Approximately 11,000 new patients per year in the United States injure their spinal cords.[11] The injuries occur secondary to falls, motor vehicle accidents, gunshot wounds, diving, and other sports-related accidents. Most victims are young males. Cervical spine injuries are the most frequent injury type (almost 50% at C4 to C7), and appropriate evaluation and immediate immobilization can prevent further deterioration. Spinal cord injury should be suspected in any patient with flaccidity (especially of the rectal sphincter), complaints of neck or back pain, maxillofacial trauma, penetrating neck trauma, or hypotension with bradycardia and warm extremities.

Early immobilization is imperative, as approximately 40% of cervical spine injuries produce neurologic damage, and 25% of this damage is caused by improper immobilization.[29] Numerous devices have been used to immobilize the cervical spine. Of these, the use of sandbags on both sides of the patient's head, which is secured with 3-inch cloth tape across the forehead to a spine board as well as the sandbags, and a Philadelphia collar have been shown most effective.[26]

Evaluation of the cervical spine requires at least a lateral radiograph. More complete evaluation may require CT scan or multiple radiographs. Interpretation of a lateral cervical film should be possible by all anesthesiologists. All seven cervical vertebrae must be visible. The following evaluation scheme from MIEMSS is useful in interpreting the lateral cervical spine.

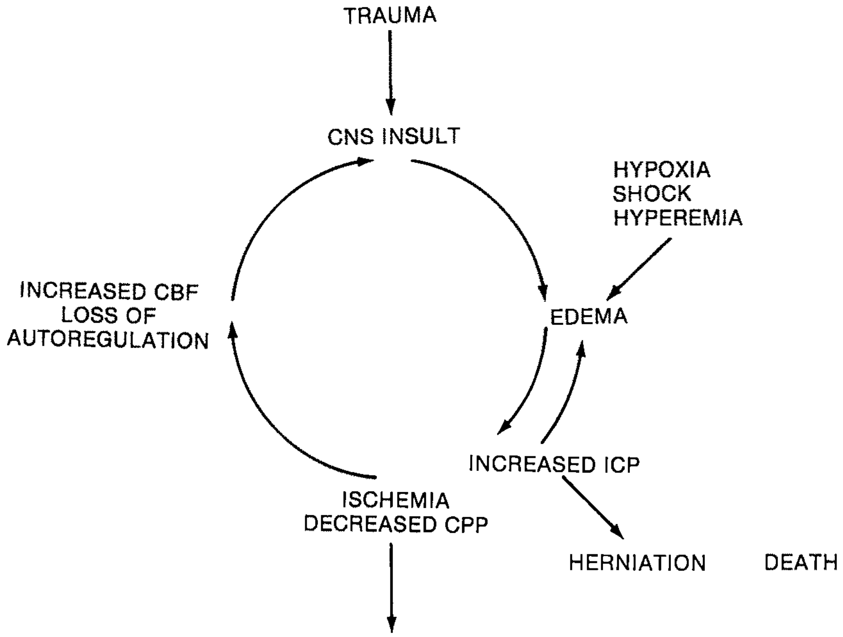

Figure 2. The viscious cycle of a head trauma related secondary brain insult. Attention to physiologic parameters can improve outcome. (*Adapted from* Cowley RA, Dunham CM: Shock Trauma/Critical Care Manual: Initital Assessment and Management. Baltimore, Aspen Publishers, © 1982; with permission.)

1. Anterior and posterior borders of cervial vertebrae should be aligned.
2. Alignment of spinolaminal lines should be evident.
3. Evidence of vertebral body, spinous process, laminae, pedicle, or odontoid fractures should be searched for.
4. Preodontoid space should be < 3 mm.
5. Normal lordosis should be present.

If the cervical spine film is suspicious or the patient reports neck pain, neurosurgical consultation is indicated. A lack of any of these abnormalities does not exclude the possibility of cervical spine injury.

Acute Spinal Cord Injury

The acute spinal cord injury presents several particular problems related to respiration, hemodynamics, and temperature regulation. These problems make the care of spinal cord injured patients exceptionally challenging to even the most experienced anesthesiologist.

Intubation may need to be performed early, as head injuries with depressed consciousness or facial injuries with bleeding and upper airway edema may leave the airway unprotected. These patients generally have full stomachs and are often hypoxic, making the need for supplemental oxygen imperative. Oxygen can be administered by a bag and face mask while maintaining cricoid pressure. Cricoid pressure will prevent gastric insufflation and passive regurgitation.[31] A recent review of several thousand patients with spinal cord injuries revealed that oral intubation is both safe and effective.[16] Oral intubation should be performed with manual in-line axial stabilization. The vast majority of patients can be intubated in the neutral position. Alternative means of intubating these patients are blind nasal intubation and fiberoptic-guided nasal or oral intubation. In general the nasal route should be avoided, particularly in those patients with facial injury or possible skull fracture.

Impaired ventilation may result from a compromised airway, chest wall injury, or the spinal cord injury itself. With injuries below the C6 to C7 level, the diaphragm will remain intact. However, intercostal paralysis may produce paradoxical breathing, loss of ability to cough, and difficulty with secretions.[11] Although the patient with a C6 to C7 injury may be able to ventilate effectively, those with injury at or above C5 are markedly impaired secondary to diaphragmatic paralysis.

A state of shock may occur from spinal cord injury. The mechanism is a sudden loss of sympathetic innervation. This sympathectomy manifests as hypotension and bradycardia and is usually the result of transection above the T4 level. Pooling of blood in the lower extremities takes place, and thus, elevation of the legs can be helpful. Volume resuscitation with the addition of direct vasopressors may be warranted with pulmonary artery catheter guidance. Caution should be used with fluid administration, as pulmonary edema may develop.[11]

Hypothermia is a significant problem in the patient with acute spinal cord injury. These patients have lost the ability to sweat or vasoconstrict and become poikilothermic.[28] Measures should be taken to prevent further heat loss and provide rewarming.

SPECIAL CONSIDERATIONS

Pre-existing Medical Problems

The trauma victim is by no means immune from medical problems. In fact, a pre-existing disease may be the initiating factor in the chain of events leading to injury. Patients with significant cardiovascular or neurologic disease may be more prone to trauma than the general population. Myocardial infarction, cardiac dysrhythmias, or seizure disorders can be incapacitating and, for example, prevent a person from controlling a vehicle.

Alcohol use frequently plays a role in motor vehicle accidents, falls, burns, homicide, and suicide. The incidence of violent crime related to drug abuse appears to be on the rise in many cities. As a result, more anesthetics are being administered to patients "high" on cocaine, crack, heroine, amphetamines, and others.

Although the trauma victim presents an acute problem, the anesthesiologist must consider substance abuse and concurrent medical problems in both the preanesthetic evaluation and anesthetic plan.

Full Stomach

Aspiration is a critical problem in trauma victims. Frequently, they have just eaten, have delayed gastric emptying, or have bloody pharyngeal secretions with diminished airway reflexes. Trauma victims may aspirate gastric contents, food, saliva, blood, or foreign bodies at the time of injury, shortly thereafter, or during endotracheal intubation for airway control or general anesthesia. Aspiration can occur while an endotracheal tube is in place. A cuffed endotracheal tube does not ensure security from aspiration. Patients may aspirate around the cuff, the cuff may rupture, or cuff deflation may permit passage of aspirate. In addition, emergence from anesthesia may find the patient with absent laryngeal reflexes, unable to protect the airway.

The significance of aspiration depends upon the material aspirated, quantity aspirated, and acidity of the aspirate.[22] Symptoms may be mild, with tachypnea, transient bronchospasm, and cough, or severe, with fulminant respiratory failure, pulmonary edema, and pneumonitis.[22]

Treatment is best aimed at prevention. Some of the more traditional means of prevention, e.g., delaying surgery are not applicable to the trauma patient. Pharmacologic intervention may be useful. The risks and benefits of each agent, however, must be weighed individually for each patient. Intubation of the trachea requires either an awake approach under topical anesthesia or a rapid sequence technique after preoxygenation. Preoxygenation should be by spontaneous respiration. Cricoid pressure must be maintained until endotracheal tube placement is confirmed.

SUMMARY

The multiple trauma victim taxes even experienced anesthesiologists, emergency physicians, and trauma surgeons. Appropriate care centers around organized planning, team cooperation, communication, and an appreciation of concerns relevant to all involved specialties. The preanesthetic evaluation should begin upon the arrival of the patient to the admitting area. Evaluation and initial stabilization often occur simultaneously, with particular attention given to the cardiopulmonary and neurologic systems. Anesthetic management needs to be tailored individually recognizing the type of injury, organ system involvement, and response to initial therapy. In short, impeccable assessment is the key to successful management and outcome.

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Preanesthetic Assessment of the Drug Abuse Patient

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The three most commonly abused drugs that influence anesthesia administration are alcohol, cocaine, and heroin. Cocaine and heroin are cited most frequently by medical examiners reporting to the Drug Abuse Warning Network (DAWN) of the National Institute on Drug Abuse.[7]

Because drug abuse and violence often are associated, the preanesthetic assessment of drug abuse patients often includes not only evaluation of even life threatening injury but an estimation of the organ system alteration caused by drug ingestion.

THE ALCOHOLIC PATIENT

It is estimated that 10 million Americans are addicted to alcohol. Each year alcohol is involved in more than 50% of automobile fatalities, 80% of fire-related deaths, 67% of drownings, 67% of homicides, 35% of suicides, and 85% of the deaths from liver disease each year.[15] Alcohol is the leading killer of persons aged 15 to 45.

Metabolism

Alcohol is rapidly absorbed; peak blood levels are reached in 30 to 60 minutes. Twenty-five percent of the alcohol is absorbed from the stomach and the remainder from the small intestine. Ninety percent of the alcohol is metabolized in the liver, and 10% is excreted by the lungs, skin, and kidneys. Alcohol dehydrogenase catalyzes the removal of H^{2+} ions from ethanol by reduction of nicotinamide ad-

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enine nucleotide (NAD) and thus acetaldehyde and reduced NAD are produced. Further oxidation produces acetic acid and then CO_2 and H_2O.

Alcohol is metabolized at a constant rate regardless of the amount present (a zero-order kinetic). Chronic alcoholic patients appear to tolerate alcohol better because of cellular adaptation of the central nervous system.

Large quantities of reduced NAD cause lactic acidosis, alcoholic ketoacidosis, hypoglycemia, and hyperuricemia. These reduced NAD levels cause a shift toward the production of lactate from pyruvate, and because pyruvate is no longer available for gluconeogenesis, hypoglycemia results. Common urine indicators for ketone bodies do not detect the β-hydroxybutyrate that is released, thus the degree of ketonemia is often underestimated.[20] In addition, free fatty acids are released as a direct effect of alcohol.

Chronic alcohol abuse causes proliferation of the endoplasmic reticulum of hepatocytes and increases the microsomal ethanol oxidizing system. The alcoholic, therefore, when not drinking may consume large amounts of sedatives and analgesics with little effect. The combination of alcohol and sedatives is dangerous because of the competition for a common metabolic path.

Older people (>65 years) are more susceptible to alcohol toxicity. The peak blood level is increased by 20% because of a diminished volume of distribution.

Preanesthetic Assessment

Diseases related to alcohol abuse may affect any organ system. The principal effects are listed in Table 1.

Central Nervous System. Denial of the amount of alcohol consumption is an important part of the alcoholic syndrome. An executive who acknowledges only a few daily drinks but who consumes much more may be at risk of a postoperative withdrawal seizure, especially if the surgery is extended. Frequently the alcoholic patient requires an early morning drink to reverse signs of withdrawal (tremor, flushing, nausea, vomiting). The physician's request to refrain from oral alcohol intake may well be ignored by the patient.

The efficacy of prescribing phenytoin to prevent alcohol-related seizures has not been established. Rather, alcohol can be administered intravenously as part of fluid replacement.

Delirium tremens, characterized by agitation, confusion, hallucinations, disorientation, fever, and hyperactivity of the autonomic nervous system, occurs in 5% of alcoholics within 3 to 5 days of abstinence. It is associated with a 5% to 15% mortality, and death is usually caused by complicating factors such as pneumonia or gastrointestinal hemorrhage. Death solely from delirium tremens is caused by hypopyrexia and hypotension with cardiovascular collapse. About 95% of cases of delirium tremens resolve spontaneously in 72 hours.

Although clinical experience has suggested that the acutely in-

Table 1. *Alcohol-Related Illneses*

ORGAN	SYNDROMES CAUSED BY TOXIC EFFECTS	SYNDROMES CAUSED BY NUTRITIONAL EFFECTS
Brain	Alcoholic dementia (cortical atrophy) Withdrawal seizures Delirium tremens	Wernicke-Korsakoff syndrome Cerebellar degeneration Central pontine myelinosis (secondary to electrolyte changes during therapy)
Nerves		Peripheral polyneuropathy (thiamine deficiency)
Heart	Alcoholic cardiomyopathy	Beriberi heart disease (thiamine deficiency)
Blood	Leukopenia, anemia, thrombocytopenia, coagulopathies	Macrocytic hyperchromic anemia (folic acid deficiency)
Gastrointestinal tract	Acute and chronic gastritis Acute and chronic pancreatitis Carcinoma of the head and neck and of the esophagus	Malabsorption syndrome (folic acid deficiency)
Liver	Fatty degeneration Acute hepatits	Laennec's cirrhosis
Metabolic	Hyperlipidemia Hyperuricemia (exacerbation of gout) Hypoglycemia	

Adapted from Kissin B: Alcohol abuse and alcohol-related illness. *In* Wyngaarden JB, Smith LH Jr (eds): Cecil Textbook of Medicine, ed 18. Philadelphia, WB Saunders, 1985, p 55.

toxicated individual requires less anesthetic and the chronic imbiber requires more, a recent study has not confirmed these suggestions.[36] No pharmacokinetic or pharmacodynamic differences were found in chronic alcoholic patients who were given pentothal to the point of electroencephalogram depression when compared to control patients. Also the dosage ranges required to achieve this point were similar.[36] Nevertheless, if possible, surgery should be postponed in an acutely intoxicated patient for a few hours.

Cardiorespiratory System. Alcoholic patients appear to be more sensitive to the cardiovascular depressant effects of anesthetics. Hypertension also has been associated with chronic alcoholism, as has peripheral vascular disease. Tolerance to anesthesia after chronic alcohol exposure, however, is not accompanied by any lesser degree of cardiovascular depression when anesthetics are administered.[38] With intravenous anesthetic agents there is generally a decrease in the amount required with increasing age to achieve anesthesia, but this does not apply to alcoholic patients. This is important, as older patients may be less able to tolerate the depressant effects of the required dosage. These effects are at cellular level, but pharmacokinetic changes can also occur. Approximately 20% of alcoholic patients have chronic obstructive pulmonary disease caused by tobacco smoking. Appropriate anaesthetic evaluation may include chest radiographs, pulmonary function tests, and arterial blood gas analyses.

Blood. The major cause of coagulopathy in the alcoholic patient is liver disease. Parenchymal involvement may lead to deficiencies of all of the proteins involved in hemostasis. Advanced liver disease also may be associated with portal hypertension and thrombocytopenia secondary to hypersplenism. Patients with extensive liver disease also are prone to disseminated intravascular coagulopathy, as the liver is not able to clear the activated clotting factors from the plasma. No specific therapy exists for bleeding caused by liver disease, and the treatment is mainly supportive. Parenteral administration of vitamin K for 3 days is only beneficial if the liver disease is so severe that little, if any, synthetic function remains. If acute bleeding develops, administration of platelets, fresh frozen plasma, and cryoprecipitate is the best treatment.[24]

The inebriated trauma patient who is in shock may sustain a bleeding diathesis for many reasons. Poor tissue perfusion, stagnant blood flow, damage to the endothelial surface of the vasculature, decreased liver perfusion with subsequent diminished production of coagulation proteins, and decreased clearance of activated factors all contribute to the diffuse intravascular thrombosis and bleeding that may occur. Treatment involves correction of the syndrome's underlying cause. Fresh frozen plasma may help to reestablish the balance between clot formation and lysis, because it contains both clotting factors and inhibitors.

In evaluating the alcoholic patient for coagulopathy the anesthesiologist should look for stigmata of chronic liver failure, such as spider angiomata, palmar erythema, jaundice, ascites, Dupuytren's contracture, gynecomastia, or enlarged parotid or lacrimal glands. The anesthesiologist also should question the patient about excessive bleeding with prior procedures, epistaxis, or easy bruising.

The basic laboratory test starts with a complete blood count. The prothrombin time measures the extrinsic pathways and the partial thromboplastin time the intrinsic pathways. However, activity loss of the various coagulation factors must approach 60% to 80% to prolong the prothrombin or partial thromboplastin times. Patients with liver disease may have normal values with moderate depletion of factors and still be at risk for surgical bleeding.[26]

In patients with coagulopathies, regional anesthetic techniques are not recommended unless unavoidable or unless the bleeding time can be returned to close to normal. No specific general anesthetic technique is indicated; however, prolonged nitrous oxide administration has been associated with decreased white blood cell and bone marrow function. One unpublished study has indicated that emergency surgery and anesthesia are not associated with worsening abnormalities of liver enzymes.[39]

THE COCAINE-DEPENDENT PATIENT

Cocaine is a major public health threat with social, economic, and political overtones. The high abuse potential coupled with few recognized medical uses classifies this as a schedule D drug. The recent surge in abuse began in the 1970s; the National Institute of Drug Abuse reported a fivefold increase in hospital admissions for cocaine-related problems during that decade. A decreased street price, easier availability, and increased purity have contributed to its use by more than 30 million Americans.[10] Of the five million regular American users, young white men between the ages of 18 and 25 comprise the largest subgroup.

The impact of this recent surge in abuse is best demonstrated by a study of motor vehicle fatalities in New York City between 1984 and 1987 that found at least one in four drivers who were killed had documented use of cocaine within 48 hours of death.[22] Another chilling fact is that deaths occur after all routes of administration of cocaine. Clearly, the severity of danger of this drug is not yet recognized by the public. Table 2 lists some of the many street names of cocaine.

Characterization of the Alkaloid

Cocaine is benzoylmethylecgonine, ($C_{17}H_{21}NO_4$), and has a molecular weight of 339.81. The amino alcohol base, ecgonine, is chemically related to atropine. The alkaloid is extracted from the leaves of *Erythroxylon coca* and is dissolved in hydrochloric acid to form a salt. The hydrochloride form exists as water soluble crystals, granules, or white powder, which decompose at 195°C. This relatively pure cocaine is then adulterated (i.e., cut or stepped on) with various impurities, including sugars such as mannitol, lactulose, glucose, and inositol, or local anesthetics such as lidocaine, benzocaine, procaine, and tetracaine. Additional often-found impurities are other drugs, including caffeine, heroin, phencyclidine, quinine, and amphetamines. The purity of street samples varies from 15% to 60% cocaine.[37]

The alkaloid form (known as free-base, crack, or rock) exists as a

Table 2. *English and Spanish Street Names for Cocaine*

English	Bernice, bernies, blow, C, Carry Nation, Crock, Cecil, coconut, coke, Corrine, dream, flake, friskypowder, girl, gold dust, heaven dust, incentive, jam, love drug, moon dust, nose candy, paradise, rock, sniff, snow, star dust, toot, uptown, white snuff
Spanish	coca, nieve, perico, polvito

colorless, odorless transparent crystal that is soluble in alcohol, acetone, ether, and oils. It is commonly re-extracted from the hydrochloride salt with baking soda, then mixed with a solvent such as ether. Contaminants may persist, even after such repurification. In this form, the crystals will melt at 98°C and vaporize at higher temperatures. Since it is not destroyed by heating, the free base alkaloid may be added to cigarettes, or marijuana, and smoked.

Pharmacokinetics. Virtually every mode of drug administration has been tried with cocaine. The first documented use of cocaine was topical application of saliva directly into a wound by an attendant who was chewing coca leaves. Intramuscular, intravenous, intrathecal, and gastrointestinal (both intentional and by "body-packing" for smuggling) methods of administration have all occurred. Topical administration with absorption through the mucous membranes gives various rates of onset and durations of action. Simple application of the water soluble hydrochloride using powder or solution can be made to conjunctival membranes, nasal mucosa (as in snorting), oral mucosa (both sublingual and buccal), rectal mucosa, and vaginal mucosa. Sustained use is invariably associated with mucosal inflammation and sloughing.

Mucous membrane absorption is slow, with delayed onset and sustained duration of action. Snorting can produce euphoria that lasts as long as 1 to 1.5 hours. Other routes have variously quicker onset rates with shorter durations. The inhalation route provides the equivalent dose as the intravenous route and comparable onset and duration. A single inhalation or "hit" of the free alkaloid (base) form delivers about 67 mg of free base, with euphoria lasting about 20 minutes. The only problem with this route is that it requires deep, forced, prolonged inspiration, with Valsalva's maneuver. The redistribution of physiologic forces by this mode of administration can lead to many different complications, including syncope, cerebral hemorrhage, and aortic rupture.[10]

After administration, the drug may persist in the urine of an adult for 24 to 36 hours. Plasma and liver cholinesterases metabolize cocaine; most of the breakdown occurs in the liver within the first 2 hours of administration. The principal metabolites are rapidly excreted in the urine. In patients who have decreased plasma cholinesterase activity, such as those with severe liver disease or congenital cholinesterase deficiency, and in the geriatric population, pregnant women, and fetuses, there is the risk of sudden death from overdose.

Pharmacodynamics. Cocaine acts primarily as a local anesthetic by blocking conduction in nerve fibers. The rapid increase in sodium permeability normally seen during depolarization is prevented by cocaine. In addition, cocaine alters synaptic transmission. It blocks the re-uptake of norepinephrine and dopamine by nerve terminals, causing excess catecholamines to accumulate at the postsynaptic receptor sites. The sympathetic nervous system is mostly stimulated, causing local vasoconstriction, an acute rise in arterial pressure, tachycardia, ventricular dysrhythmias, seizures, mydriasis, hyperglycemia, and hyperthermia. The dopaminergic effects result mostly in euphoria.[11]

There is also a direct action causing vascular smooth muscle contraction, which is independent of the sympathetic nervous system and is not affected by phentolamine or prazoxin. This activity is inhibited by diltiazem.

Life-Threatening Side Effects

The physiologic derangements caused by cocaine result in many life-threatening side effects; these include cardiac dysrhythmias, acute myocardial infraction, cerebrovascular accidents, seizures, acute pulmonary edema, hyperthermia, gastrointestinal ischemia, spontaneous abortion, abruptio placenta, and acute rhabdomyolysis.[1, 2, 9, 19]

The hyperthermia seen with cocaine abuse is attributed to generalized vasoconstriction coupled with increased muscle activity.[35] Not only is there a loss of heat dissipation but there appears also to be a change in the thermoregulatory center, thus permitting acute rises in temperature to persist. This change is possible because of local vasoconstriction at the thermoregulatory center itself.

Occasional deaths occur from accidental rupture of swallowed cocaine packages carried by smugglers ("body packers"). This, as well as oral discharge of cocaine, can easily induce norepinephrine stimulation of intestinal vasculature followed by reduced blood flow, which then results in bowel ischemia. This diagnosis must be considered in any cocaine abuser complaining of abdominal pain. A marked leukocytosis in such a patient may indicate gangrenous bowel.

The rhabdomyolysis associated with free-base cocaine overdose and intravenous abuse has resulted in nonoliguric renal failure and in one patient, hyperkalemic death.[16, 17, 32]

Chronic Cocaine Abuse and Associated Medical Problems

Many chronic minor medical problems plague cocaine abusers and should serve as clues to the presence of chronic cocaine usage. The abuser often reports fatigue, increased hunger, and mental depression but appears nervous and agitated, and exhibits paranoid thinking. A neurologic exam may only show heightened reflexes, but there often is a decreased sense of smell. Cerebral vasculitis has been reported.[18]

A physical examination may reveal nasal septal atrophy; necrosis and septal perforation may even be present. Nasal verrucae are another finding associated with cocaine abuse by snorting. Generalized noncarious tooth destruction may result from loss of both dentin and enamel as the mineral becomes dissolved by the hydrochloride solution applied to the gingiva. There is a resting tachycardia, and the patient may show electrocardiograph changes consistent with findings of myocardial ischemia. The chest radiograph may be abnormal, revealing atelectasis or localized parenchymal opacification.[8] Fever, transient pulmonary infiltrates, eosinophilia, pruritis, elevated immunoglobulin E levels, and bronchospasm following cocaine inhalation have been dubbed "crack lung".[9, 19]

The use of cutting agents in drug mixtures that are injected may result in pulmonic foreign body granulomatosis.[4] Chronic use results in a loss of diffusing capacity and decreased compliance.[3]

Many cardiac abnormalities have been linked to cocaine abuse, including ischemic events, sudden dysrhythmias, myocarditis, cardiomyopathy, and valvular heart disease.[17] Cocaine induces coronary artery vasospasm by a direct effect and by an effect of catecholamine. The most common rhythm changes are serious tachycardia and ventricular dysrhythmias. In a study of 21 patients undergoing cocaine withdrawal, 40% of the cocaine abusers had ST-T elevation on 24-hour electrocardiographic monitoring. In some patients these ischemic changes did not resolve for up to 6 weeks.[30]

Apart from the euphoria associated with cocaine ingestion, several other central nervous system complications have been described.[29] Headache may indicate internal hemorrhage.[31] Seizures, which may be induced by a single dose, may be caused by hyperpyrexia combined with neurotransmitter intensification or may be secondary to a myocardial event.[23] Thalamomesencephalic strokes, ischemic as well as hemorrhagic, have been described in young people after cocaine use.[25] Other neuroleptic diseases include anterior spinal artery syndrome, lateral medullary syndrome, transient ischemic attacks in the middle cerebral artery and vertebrobasilar artery areas, and partial motor seizures.[29]

Preanesthetic Preparation

At the time of the initial examination of the patient, the timing and administration route of all drugs in the last 24 hours and the length of addiction should be recorded. The anesthesiologist should try to correlate signs of recent drug use with physical findings. Pinpoint pupils usually indicate recent narcotic administration, whereas widely dilated pupils indicate use of sympathomimetic drugs (amphetamine or cocaine).

Hypertension, which may be severe, should be treated with phentolamine. Propranolol may help to reduce tachycardia. An adequate blood pressure should not be used as an indicator of normovolemia. Rather, a careful clinical assessment of blood and fluid loss should be made and appropriate replacement given.

The mechanism underlying cocaine euphoria is not well understood. Dopaminergic antagonists or depletors, such as reserpine, methyldopa, the phenothiazines (haldol, droperidol, and metoclopramide), can block most of the euphoric effects. Use of these drugs during anesthesia in an addicted patient may cause delayed awakening, general fatigue, lassitude, hyperphagia, and depression postoperatively. The use of phenothiazine as premedication is probably ill advised, and neuroleptic anesthesia is also a poor choice.

Intravenous diazepam may help in the acute management of seizures, but prophlylactic anticonvulsant medications have not been shown to be effective in preventing seizures.

HEROIN

Heroin, or diacetylmorphine, when injected, produces a warm flushing of the skin, and sensations in the lower abdomen that have been described by addicts as similar in intensity and quality to a sexual orgasm. This "rush" or "kick" lasts about 45 seconds. Heroin also may be injected subcutaneously (skin popping), which delays its onset of action.

Compared with morphine, heroin has been described as a more potent analgesic that causes less nausea, vomiting, and constipation. Milligram for milligram, it is approximately 2.5 times as potent as morphine, and 25 to 30 times as potent as meperidine. One milligram of heroin is equal in analgesic activity to about 100 mg fentanyl.[13] The duration of action is approximately 2 to 4 hours.

The typical amount of heroin sold (bag) is approximately 5 to 10 mg heroin cut with about 90 mg of quinine, talc, or starch.

Heroin probably is the most commonly abused intravenously administered drug. Commonly used street names of heroin are listed in Table 3. Many of the problems associated with heroin are caused more by the complications of intravenous injection than by the drug itself. Again, all organ systems may be affected.[6]

Respiratory Assessment

Opioids decrease cough reflexes and mucociliary clearance, which decrease peak expiratory flow. As most addicts also smoke cigarettes, the effect is similar to chronic bronchitis, i.e., increased partial pressure of carbon dioxide, arterial ($Paco_2$), decreased partial pressure of oxygen, arterial (Pao_2), and pulmonary hypertension. Induction by an inhalation technique is prolonged in these patients.

Pulmonary function tests should be performed routinely before elective surgery. If intubation in the awake state is deemed necessary in the emergent situation, an estimation of forced expiratory volume can be obtained by attaching a Wright spirometer to the end of the tube and measuring vital capacity. A chest radiograph is indicated in all patients, and arterial blood gas analyses provide useful baseline

Table 3. *English and Spanish Street Names for Heroin*

English
  bomb, boy, brother, brown sugar, cat, chick, Chinese red, crap, dogie, doojee, dope, duji, H, Har y, horse, junk, Mexican mud, scag, scat, smack, stuff, thing
Spanish
  blanco, caballo, cargo, chiva, la cosa, la duna, gato, golpe, H, manteca, stufa

Data from National Institute on Drug Abuse.

information. During a "trip" the patient is often stuporous, a condition commonly accompanied by hypoventilation and predisposition to aspiration.

Cardiac Assessment

Intravenous drug addicts often have signs and symptoms of cardiovascular disease. Endocarditis is common. Typically the tricuspid valve becomes involved before the mitral or aortic valves.

Electrocardiographic abnormalities are common, ranging from a prolonged Q-T interval to electrocardiographic evidence of myocardial infarction.[28]

Low diastolic blood pressure is common among addicts and is thought to be caused by decreased sympathetic tone, which also decreases systemic vascular resistance.[34]

Liver Function

Most heroin addicts have significant liver abnormalities manifest by elevations in serum glutamic-oxaloacetic transaminase, serum glutamic-pyruvic transaminase, and alkaline phosphatase. Pathologic changes range from mild lymphocytic infiltration of the portal triads to severe fibrous tissue bridging between the triads. The causes of these changes are controversial. Explanations such as malnutrition and chronic toxic reaction have been postulated.[14, 21]

A history of hepatitis can be elicited from 50% of addicts, with one third having more than one episode. Hepatitis B surface antigen (HBsAg), the serologic marker of the virus, is an indicator of infection. HBsAg is reported to be present in 2.2% of addicts, four times the incidence in the general US population.

Cirrhosis of the liver is also prevalent, especially in young abusers.[12] The onset of cirrhosis is earlier for addicts as compared with alcoholics. The reason for this is unclear. Talc and other contaminants that pass through the pulmonary system through arteriovenous shunts are often found in the liver parenchyma and may cause granulomatous disease.

As with alcoholics, biochemical manifestation of liver disease may not be present until much of the reserve has been exhausted.

Anesthetic agents and neuromuscular blocking agents that are metabolized by the liver (e.g., opioids, pancuronium, halothane) should be avoided.[5] Isoflurane appears to be the agent of choice, because less than 1% is metabolized by the liver, even in the presence of hepatic enzyme induction. Similarly, atracurium, which depends on Hoffman elimination, is useful.

Musculoskeletal Abnormalities. Ischemic necrosis of muscles and skin may result when the heroin addict is comatose and pressure over bony prominences is increased. Edema formation and the crush syndrome results.[12]

Fluid shift to the injured area can be sufficient to cause hypovolemia, and with muscle tissue necrosis, myoglobinemia may result in obstruction of the renal tubules and renal failure.[27] Rhabdomyolysis has been described and is believed to be caused by a contaminant of the heroin.[21, 33] The urine is dark. Laboratory tests may detect myoglobin and free hemoglobin. The urine should be alkalinized with sodium bicarbonate (approximately 1 mg/kg). To prevent precipitation in renal tubules, a loop diuretic such as furosemide (20 mg) should be given.

Osteomyelitis is not uncommon in these patients. The lumbar vertebrae seem to be particularly at risk.[14] The reason for this predisposition is not well understood. If a regional anesthetic is to be considered in the addicted patient, a radiograph of the vertebral column should be obtained to rule out involvement.

Central Nervous System Assessment

Central and peripheral neurologic lesions may be caused by anaerobic phenomenon from vegetation or heart valves. Documentation of lesions is necessary to prevent confusion of new postoperative onset changes. Tetanus has been reported in heroin addicts who use the skin popping technique.[14, 21, 33] The predisposition to this is believed to result from the use of quinine, which lowers the redox potential of tissues at the injection site, and in turn predisposes to anaerobic contamination.

Heroin Withdrawal

Reactions to heroin withdrawal begin 24 to 36 hours after the last opiate injection. The first signs are often perspiration and rhinorrhea, followed by tremors, muscle twitching, hot and cold flashes, and systemic aching. Later symptoms include fever, vomiting, diarrhea, spontaneous ejaculation, and orgasm. Involuntary muscle spasms produce kicking movements, and the patient often assumes the fetal position. Frank pulmonary edema may develop.

Before induction of anesthesia, the symptoms of withdrawal must be ameliorated. This can be accomplished with methadone. Methadone, 20 mg, blocks the symptoms of severe withdrawal in almost all addicts. During a long surgical procedure withdrawal symptoms may develop. Signs include increased airway pressure and pulmonary edema, fever, tachycardia, hypotension, and muscle twitching suggestive of focal or general seizures.

Anesthetic Complications

A low tolerance of pain is common. Topical application of preservative-added sterile water (which contains benzyl alcohol 0.9% vol/vol) provides anesthesia to the skin before the insertion of intravenous cannulae. The advantage of this technique over lidocaine infiltration is the lack of any burning sensation. Because of repeated patient use veins are thrombosed or sclerotic, and establishment of intravenous access may be extremely difficult. Central cannulation is preferable to unreliable peripheral venous access. Using a mask induction may be better than repeated unsuccessful affects at entering a vein.

Induction of anesthesia with thiopental probably is preferable to the use of etomidate or ketamine. Etomidate has been implicated in suppression of adrenal cortical function. In the presence of narcotics, ketamine has a prolonged action and may increase the existence of confusional states.

Heroin-Cocaine Combination

A "speedball" is a line of cocaine mixed with one or two bags of heroin. The mixture is placed in a teaspoon and heated to form a slurry that is then injected. A euphoric state is induced that lasts approximately 30 minutes; this is followed by a period of stupor lasting 2 to 4 hours. The pharmacodynamic effect of the combination of these two drugs may be differentiated by the presence of sweaty hands in the cocaine-predominant phase.

To plan an anesthetic induction, it is imperative to ascertain as best as possible the time of slurry injection. Induction during the stimulation (cocaine-predominant) phase may require high doses of anesthetic, whereas a low dose will suffice during the heroin-predominant stage.

Protection of Medical Personnel

Drug addicts are at increased risk of contracting hepatitis B and the acquired immunodeficiency syndrome (AIDS). The pathogenesis and symptoms of these entities are discussed by Gotta elsewhere in this issue, as are the appropriate precautions that should be taken by all medical personnel caring for patients with these diseases.

SUMMARY

Drug addiction remains a major health hazard in our society, especially in urban areas. Drug combinations appear to be increasing as well. Toxicity has far-reaching effects on all organ systems, and the anesthesiologist caring for these patients must have an understanding not only of the adverse effects of illegal drugs but also the interactions that may occur with anesthetic agents so that appropriate adjustments may be made.

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Preanesthetic Assessment of the Pain Patient

*Jon Dean Samuels, MD**

Pain has been a part of the human condition since the origin of man. Its causes have been many and varied: disease, pestilence, wars, and childbirth. Man has struggled to understand the causes of pain and in so doing devise means to remove afflictions and ease suffering. Early efforts, shrouded with superstition and religious mysticism, were largely unsuccessful. In 1817, Frederick Serturner, an apothecary's assistant, isolated the active alkaloid of opium, morphine, which he named after the Greek god of dreams, Morpheus.[52] When he published his work in a paper entitled "Principium Somniferum," a new era had begun in which the scientific basis for the treatment of pain became possible.

Because of its subjective nature, it is often difficult to describe the nature, intensity, and location of pain. The International Association for the Study of Pain defines pain as "an unpleasant sensory or emotional experience associated with actual or potential tissue damage or described in terms of such damage".[40]

In the United States pain is the number one health complaint. Annually, more than 700 million work days are lost from chronic pain, which, when added to health care costs, total more than $60 billion.[11,60] The most common locations of pain are the head, back, and joints. These conditions are usually treated symptomatically. In most cases it is the pain and not the condition per se that incapacitates the patient. Inadequately treated pain syndromes burden patients and their families with severe emotional and psychologic damage.[11]

The field of pain management is undergoing unprecedented growth.[15] In most US hospitals a pain service has been established or is being planned. The number of residency training positions in pain, however, is limited,[15] and thus many pain practitioners learn on the job. This article is intended as a quick reference for these physicians

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involved in daily pain care. For more information the reader is referred to several comprehensive texts on pain management.[17, 39, 46]

PAIN PATHWAYS: THE GATE THEORY OF PAIN

Pain usually arises as a result of tissue injury. Its survival value is that it produces sufficient arousal and distress to stimulate avoidance behavior from noxious stimuli.[36] Afferent pain sensations originate in the periphery through nociceptors, which are free nerve endings. They are classified as high threshold mechanoceptors (HTMs) and polymodal nociceptors (PMNs). HTMs have thinly myelinated axons, conduct in the A-delta range (diameter 1–5 μm, carry impulses at 6–30 m/s), are triggered by intense mechanical stimuli (surgical incision), and are perceived quickly as well-localized "pricking" pain.[13] PMNs, the majority of nociceptors, have unmyelinated axons, conduct in the C range (diameter 0.5–2 μm, carry impulses at 0.5–2 m/s), are triggered by intense mechanical, thermal, and chemical stimuli, and are perceived slowly as poorly localized "burning" pain.[13] Nociceptors are most densely packed in the skin and mucosae as well as joint surfaces, the fascia, the peritoneum, parietal serosae, the arterial walls, and the meninges. They are sparsely packed within most deep tissues. Nociceptive-specific fibers are absent in the gut,[6] and pain perception elicited by spasm, stretching, or inflammation is transported by multipurpose afferents: viscerosomatic convergence occurs, resulting in referral of pain to cutaneous territories occupied by the same spinothalamic afferents P34N. Intraabdominal, intrathoracic and pelvic visceral pain perception is conveyed by sympathetic afferents.[34] Cardiac pain is probably conveyed by multipurpose afferents that undergo viscerosomatic convergence.[37] Brain tissue has no nociceptive fibers.[6]

Painful stimuli trigger efferent traffic along peripheral nerves. The network of distal branches to adjacent blood vessels, hair follicles, sweat glands, and mast cells; proximal branches to the paravertebral sympathetic chain (T2–L2); and rami communicates to viscera triggers vasomotor, autonomic, and visceral changes independent of spinal function.[34] A local sensitization or hyperalgesic state may be caused by repeated tissue injury; 5-hydroxytryptamine (serotinin), histamine, bradykinin, and prostaglandins are probably involved in this.[57] The therapeutic mechanism of action of nonsteroidal anti-inflammatory drugs, aspirin (cyclo-oxygenase inhibition), and corticosteroids (phospholipase A_2 inhibition) may be partially due to interference in release of endogenous mediators of tissue damage.[57] Afferent peripheral nerves pass through the dorsal root ganglion (Fig. 1), enter the dorsal root entry zone after large myelinated fibers are segregated medially, and bifurcate, sending one branch rostrally in the dorsal column and the other branch into the substantia gelatinosa where it undergoes branching and synapses with nociceptive specific neurons (NS) and wide dynamic range neurons (WDR).[62] Substance P is released in the

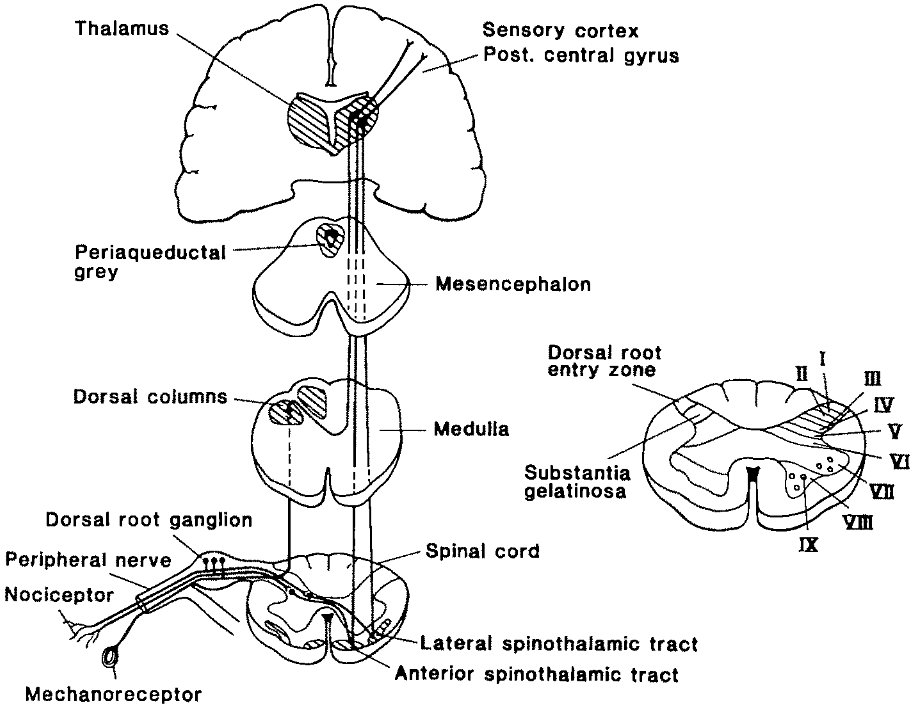

Figure 1. Afferent pain pathways (*From* Raj PP (ed): Practical Management of Pain. Chicago, Year Book, 1986, p 56; with permission.)

dorsal horn and activates pain projection neurons.[57] Spinal opioids may work by blocking the release of substance P.[21] The negative neurotransmitters glycine and gamma-amino butyric acid may also act in the dorsal horn.[21] Afferent pain messages travel along both the neospinothalamic tract (NST) and the paleospinothalamic tract (PST). The lateral NST projects into the ventral posterolateral nucleus of the thalamus and is involved in discriminating the duration, location, and intensity of pain.[8] The medial PST projects into the medial thalamic nuclei and mesencephalic reticular formation and is involved in creating the arousal, suffering, and generalized autonomic responses that accompany pain. It may also be responsible for denervation dysesthesia.[56] The periacqueductal gray area (PAG) of the midbrain is rich in endogenous opioids (enkephalins) and may exert pain-inhibitory descending control by the nucleus raphe magnus (NMR) and medullary reticular formation through serotinergic fibers in the dorsolateral funiculus (DLF) to spinal cord dorsal horn cells.[8] With peripheral nerve or spinal cord transection deafferentation paid results; this may be caused by short-circuiting of action potentials (ephaptic transmission or "crosstalk") across demyelinated segments,[8] neuroma sprouts with altered calcium transport, and increased α-adrenergic receptors,[8] or impulse generation at points of membrane instability proximal to the injury.[56] For a nerve block to be successful, the drug must be directed at the site of injury or rostral to the injury.

The gate theory of pain postulates that a "gate" for pain exists in the substantia gelatinosa of the posterior spinal horn. Melzack and Wall[39] suggested that stimulation of large-diameter myelinated cutaneous afferent fibers could close the gate to information coming over smaller, pain-carrying fibers and reduce or eliminate the perception of pain. A practical application of this theory is the use of transcutaneous electrical nerve stimulation (TENS) for acute postoperative pain.[16]

ANESTHETIC ASSESSMENT OF THE PAIN PATIENT

The anesthesiologist working in pain management functions as a consultant. Therefore, patients usually already have at least a working diagnosis of their pain and in many cases extensive medical and surgical histories detailing the pain. The referring physician must send through historical information, physical examination, laboratory and roentgenographic data before the first visit to provide an indication of the pathologic conditions involved and rule out conditions that require medical or surgical intervention. Referred patients should have an understanding of the purpose of the referral and the nature of the proposed treatment.

History

At the first visit, the anesthesiologist performs a full history and physical examination. The patient's chief complaint should be analyzed methodically. A skillfully taken history, with careful analysis and interpretation of the chief complaint, frequently indicates the probable diagnosis. The examiner should intervene only to ask leading questions or redirect the discussion from extraneous information. It is important to determine certain characteristics of any report of pain:

1. Intensity: The visual analog scale (VAS) from 0 to 10 (0 = no pain, 10 = worst pain imaginable)[1] is used. Present level at rest and while moving is determined. Intensity of paroxysms, if present, is determined.
2. Quality: The examiner asks which word(s) are best applied—dull, sharp, throbbing, aching, burning, lancinating.
3. Pattern: Is the pain worse in the morning? At night? Does it awaken the patient from sleep? Does it show seasonal variation?
4. Exacerbating factors: Do movement, weight bearing, particular positions, light touch, cold, heat, emotional factors aggravate the pain?
5. Relieving factors: Do rest, particular positions, cold, or heat relieve the pain?

6. Radiation: How does the pain radiate: ipsilateral or contralateral, constant or changing, dermatomal?
7. Associated phenomenon: Do any other conditions coexist such as muscle spasms, sleep disorders, scotomata, and depression.
8. Changes in life-style induced by the painful condition: Has a change in living occurred, such as loss of job, loss of recreational activities, changes in key relationships caused by increased dependency, lowered self-esteem.
9. Emotional state: Many pain patients are depressed. Secondary gain is often present. The psychologist will formally address this issue, so only gross manifestations of psychogenic overlay should be noted, as should suicidal ideation, alcoholism, or drug addiction.
10. Analgesics: What medications does the patient actually take and what is the visual analog scale pain relief obtained?
11. Nonanalgesic medications: Chronic medications have variable effects on the pain condition. Medical conditions should be optimized by the primary physician. The use of particular medications may preclude a proposed therapy. For example, alpha-blockers are contraindicated in the therapy of hypertensive patients receiving guanethidine Bier blocks for reflex sympathetic dystrophy.[10,30]
12. Compensation cases: Is this a work-related injury? With ongoing lawsuits the response to therapy is uniformly poor.[46]

Often a diagnosis will be obvious at this point. For example, in one case a young woman had diffuse, burning pain and dysesthesias along her left lower extremity after penicillin had been injected intramuscularly in the gluteus maximus muscle. Reflex sympathetic dystrophy of the left sciatic nerve was diagnosed. In another case an elderly man developed paroxysms of lightning-like pain along his right jaw 4 weeks after an attack of shingles. Postherpetic neuralgia of the right trigeminal nerve branch V3 was diagnosed.

Physical Examination

The initial examination should be routine. The pain practitioner must be an astute clinician and should be suspicious of pre-existing diagnoses, attempt to substantiate them with objective data, and explain discrepancies. A routine anesthetic examination delineates the presence and extent of coexisting diseases and gives a functional assessment. The patient with cancer-related pain should be adequately evaluated and staged and the mechanism of pain determined (Table 1). Vital signs are checked (supine, sitting, and standing) and the presence of orthostasis or autonomic dysfunction[20, 23] noted. A problem list with physical findings and medications is generated for each disease. The presence of a major coagulopathy should be ruled out. An abbreviated neurologic examination is performed.[46] An in-depth physical examination then focuses on the painful area. Signs and symptoms

Table 1. *Causes of Cancer-Related Pain*

CAUSE	SYMPTOMS
Secondary to Direct Tumor Involvement	
Bone involvement	Localized to area, progressive increase in intensity
Neural invasion	Burning pain, hyperesthesia, dysesthesia
Brachial plexus	Radicular pain from C8–T1, paresthesias, Horner's syndrome
Lumbar plexus	Radicular pain in L1–3 (groin, thigh)
Sacral plexus	Perianal pain, bowel and bladder dysfunction
Meningeal carcinomatoses	Headache, neck stiffness, low back pain
Epidural/spinal	Pain along invaded vertebral body or neural distribution
Visceral	
Pleural	Interscapular pain to shoulder or anterior chest
Liver	Right upper quadrant abdominal pain radiating to lower ribs
Pancreatic	Midepigastric knifelike pain through to back
Secondary to Cancer-Directed Therapy	
Postsurgical	Occurs 1–2 mo after surgery
Postsurgical	Burning pain in posterior arm, axilla, chest (intercostobrachial neuritis)
Postthoracotomy	Dysesthesias over area of sensory loss (intercostal neuritis)
Phantom limb	Burning pain over stump
Postchemotherapy	
Posttherpetic neuralgia	Pain along a dermatomal distribution, common in patients older than 50 years old with depressed immune response
Toxic neuropathy (e.g., vincristine)	Dysesthesia over hands and feet
Postirradiation	Can occur 6 mo to 20 yr after irradiation Progressive motor weakness: radiation injury to adjacent luna, rib, or humerus

From Ferrer-Brechner T: *In* Raj PP (ed): Practical Management of Pain. Chicago, Year Book 1986, p 313; with permission.

are correlated with anatomic landmarks. The involved area is examined for the presence of immobility, pathologic positions, abnormal motor activity, and appearance (atrophy). Skin is checked for turgor, hyper or anhydrosis, hair loss, pitting, or hard edema. Cutaneous temperature is compared with the contralateral side. Exteroception (pain, temperature, and touch sensation) is checked with common objects, such as an alcohol swab, a pin, or a feather. Proprioception is checked, along with two-point discrimination. Muscle strength, tone, coordination, and symmetry are checked. Joints are checked for active and passive range of motion, enlargement, crepitation, ankylosis, and warmth. Signs of arterial and venous insufficiency are sought bilaterally, and arterial pulse upstroke and capillary filling are examined. The distribution of spontaneous and provoked pain are compared.

Trigger points, muscle spasm, and guarding are sought. When hypomobility exists, it is determined whether it is on an anatomic basis (contracture) or secondary to muscular spasm. Deep tendon reflexes (DTRs) are rated and checked for symmetry. The spine and paraspinous muscles are observed and palpated; if there is kyphosis or scoliosis, the degree and direction are determined. If there is torticollis, the direction, angle, and degree of reducibility are determined. If there is a nerve lesion, the cutaneous distribution will show whether it is in a peripheral nerve (Figs. 2*A* and *B*), a major trunk (Fig. 3), or higher. Lesions in mixed nerves show distributional hypesthesias, motor deficits, and absent DTRs (Fig. 4).

Provocative maneuvers are performed.[46] In carpel tunnel syndrome, tapping on the median nerve at the wrist (Tinel's sign) or extreme wrist flexion (Phalen's sign) reproduces the pain. A straight leg raise (Lasegue's sign) reproduces sciatic pain. Pain is reproduced in hip joint disease but absent in sciatica when the external malleolus of the painful extremity is placed on the opposite knee while in a supine position and the thigh is pressed downward (Patrick's sign). Tapping on the spinous processes reproduces pain in patients with facet syndrome. Radicular pain elicited when the patient bends to the same side as the complaint may indicate nerve root compression. Pain elicited when bending to the opposite side usually indicates facet or a pathologic posterior spinal segment. With nerve lesions, primitive reflexes (signs of spinal automatism) are uncovered. An example of this is great toe dorsiflexion and splaying of the toes on lateral plantar stroking (Babinski's sign).[46] In cases of transverse spinal cord lesions, after the period of spinal shock has passed, autonomic hyperreflexia (severe hypertension and tachycardia) and Riddoch's mass reflex (evacuation of the bladder and bowels, sweating, reflex erythema, pilomotor activity, priapism and posturing below the lesion) may be precipitated by various stimuli (a full bladder, surgical incision) caudad to the transection.[46] Constant pain with distinctive, poorly localized, provoked paroxysms is consistent with thalamic syndrome.[19]

ACUTE PAIN: THE ACUTE PAIN SERVICE

Acute pain is usually the result of excessive or improper exertion, trauma, or surgery.[36] It has survival value, as it promotes immobility, thus encouraging rest and healing. However, it causes suffering, decreased vital capacity (VC), and forced expiratory volume in one second (FEV_1), and if prolonged, may precipitate myofascial and autonomic glucocorticoid, catacholamine, and glucagon levels.[59] Prolonged immobility also predispose to deep venous thrombosis.[59]

Statistics from the US Department of Health and Human Services (formerly Health, Education, and Welfare [HEW]) show that almost 53 million operations are performed annually.[60] In the postoperative period, 30% of the patients have mild pain, 30% have moderate pain, and 40% have severe pain, based on analgesic requirements.[60] Tra-

(*Text continued on page 854*)

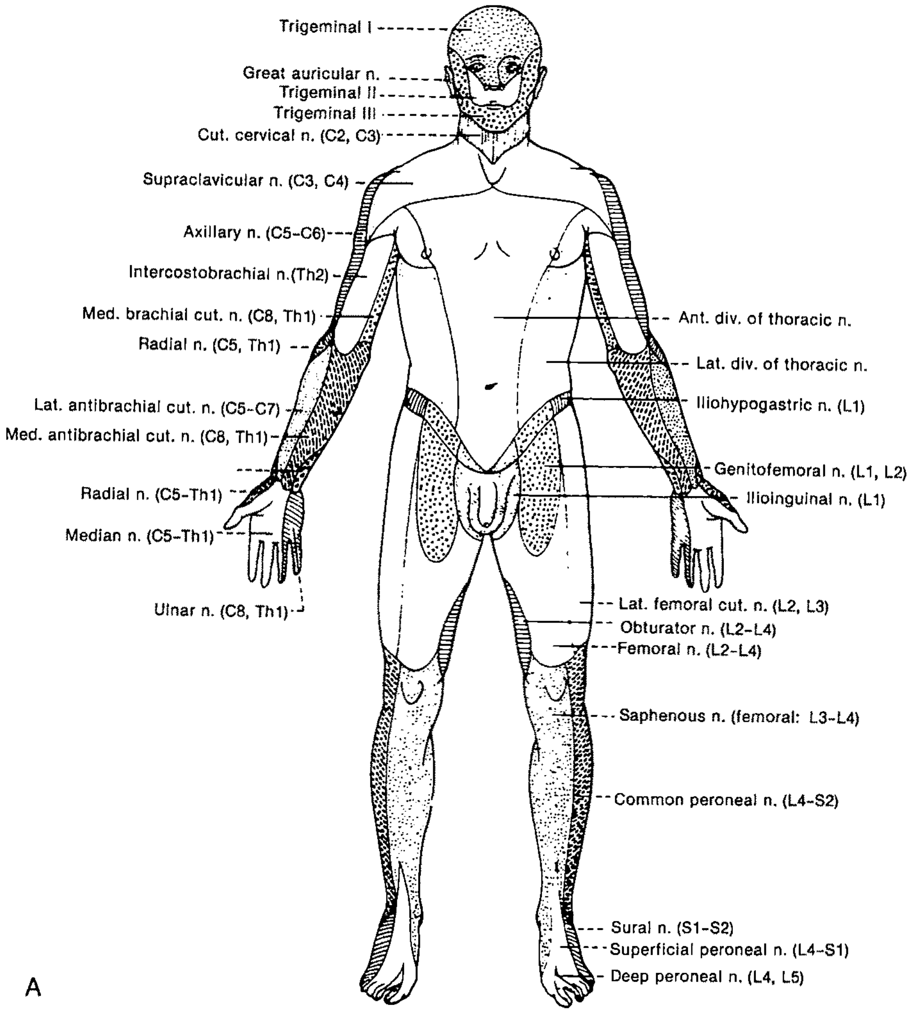
Trigeminal I
Great auricular n.
Trigeminal II
Trigeminal III
Cut. cervical n. (C2, C3)
Supraclavicular n. (C3, C4)
Axillary n. (C5-C6)
Intercostobrachial n.(Th2)
Med. brachial cut. n. (C8, Th1)
Radial n. (C5, Th1)
Lat. antibrachial cut. n. (C5-C7)
Med. antibrachial cut. n. (C8, Th1)
Radial n. (C5-Th1)
Median n. (C5-Th1)
Ulnar n. (C8, Th1)
Ant. div. of thoracic n.
Lat. div. of thoracic n.
Iliohypogastric n. (L1)
Genitofemoral n. (L1, L2)
Ilioinguinal n. (L1)
Lat. femoral cut. n. (L2, L3)
Obturator n. (L2-L4)
Femoral n. (L2-L4)
Saphenous n. (femoral: L3-L4)
Common peroneal n. (L4-S2)
Sural n. (S1-S2)
Superficial peroneal n. (L4-S1)
Deep peroneal n. (L4, L5)
A

Figure 2. *A*, Anterior cutaneous distribution of sensory nerve innervation. (*From* Baker AB, Baker LH: Clinical Neurology, vol 1. Philadelphia, JB Lippincott, 1983; with permission.)

(*Illustration contimued on opposite page*)

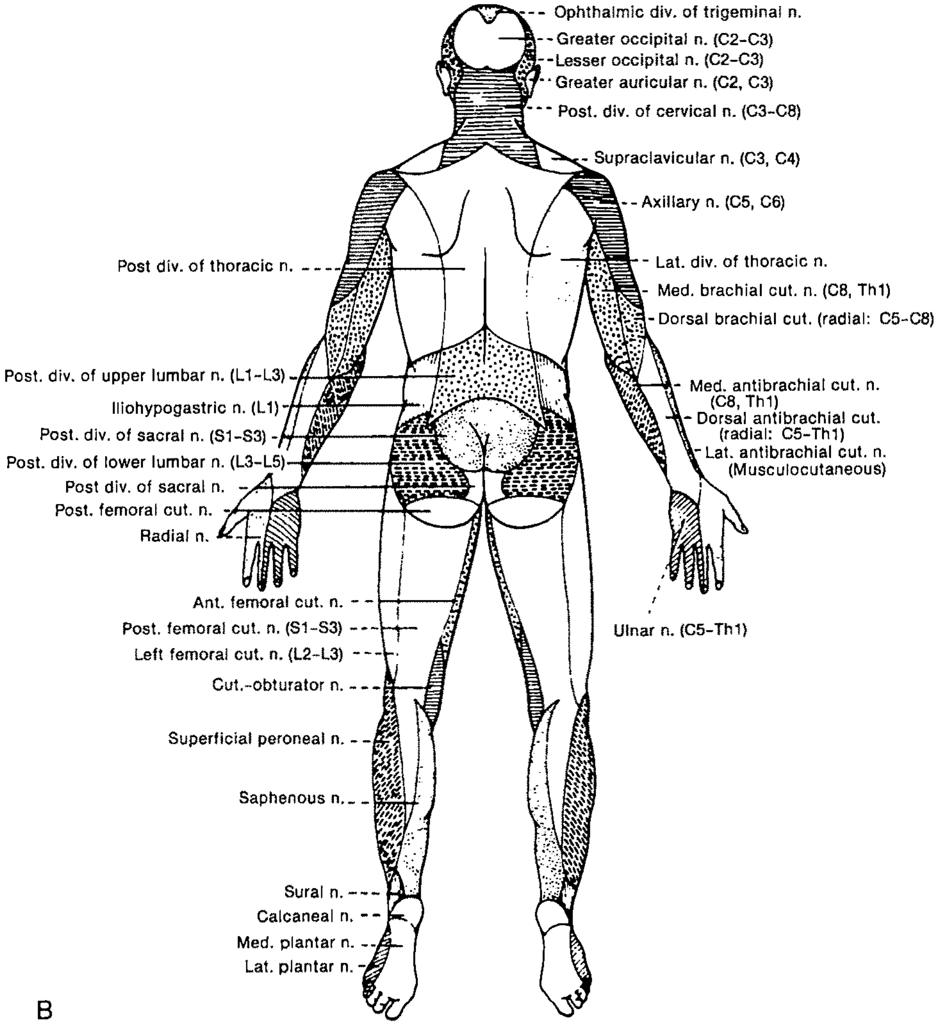
Ophthalmic div. of trigeminal n.
Greater occipital n. (C2-C3)
Lesser occipital n. (C2-C3)
Greater auricular n. (C2, C3)
Post. div. of cervical n. (C3-C8)
Supraclavicular n. (C3, C4)
Axillary n. (C5, C6)
Lat. div. of thoracic n.
Med. brachial cut. n. (C8, Th1)
Dorsal brachial cut. (radial: C5-C8)
Med. antibrachial cut. n. (C8, Th1)
Dorsal antibrachial cut. (radial: C5-Th1)
Lat. antibrachial cut. n. (Musculocutaneous)
Ulnar n. (C5-Th1)
Post div. of thoracic n.
Post. div. of upper lumbar n. (L1-L3)
Iliohypogastric n. (L1)
Post. div. of sacral n. (S1-S3)
Post. div. of lower lumbar n. (L3-L5)
Post div. of sacral n.
Post. femoral cut. n.
Radial n.
Ant. femoral cut. n.
Post. femoral cut. n. (S1-S3)
Left femoral cut. n. (L2-L3)
Cut.-obturator n.
Superficial peroneal n.
Saphenous n.
Sural n.
Calcaneal n.
Med. plantar n.
Lat. plantar n.
B

Figure 2 (*Continued*). *B*, Posterior cutaneous distribution of sensory nerve innervation. (*From* Baker AB, Baker LH: Clinical Neurology, vol 1. Philadelphia, JB Lippincott, 1983; with permission.)

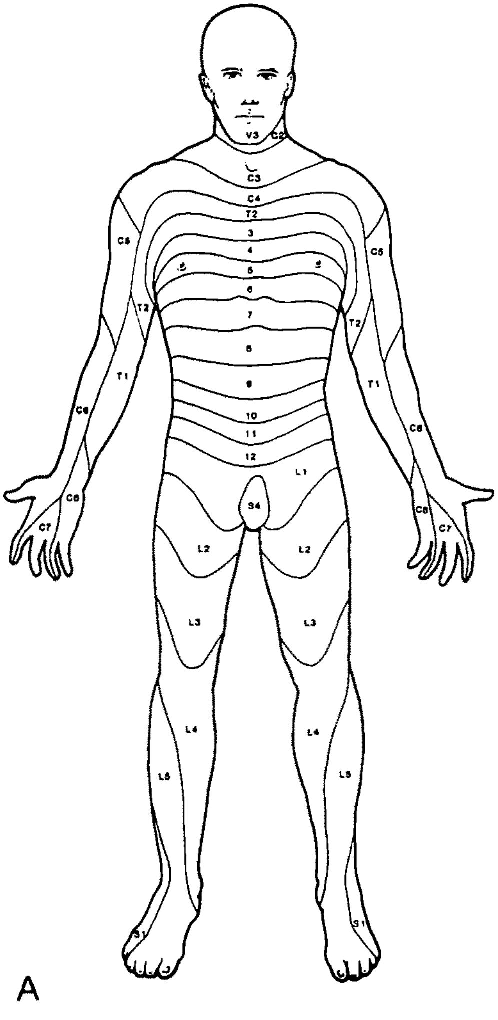

Figure 3. *A*, Anterior dermatomes of the body. (*From* Baker AB, Baker LH: Clinical Neurology, vol 1. Philadelphia, JB Lippincott, 1983; with permission.)

(Illustration continued on opposite page)

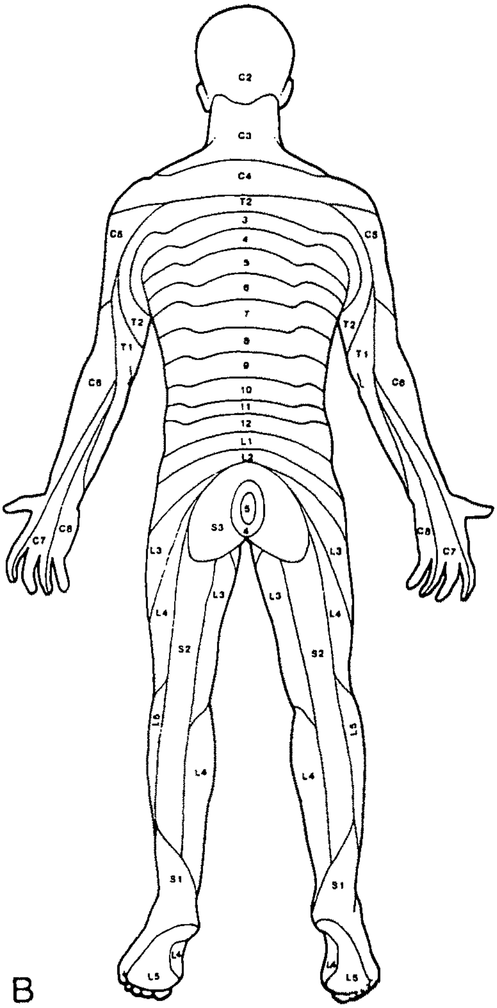

Figure 3 (*Continued*). *B*, Posterior dermatomes of the body. (*From* Baker AB, Baker LH: Clinical Neurology, vol 1. Philadelphia, JB Lippincott, 1983; with permission.)

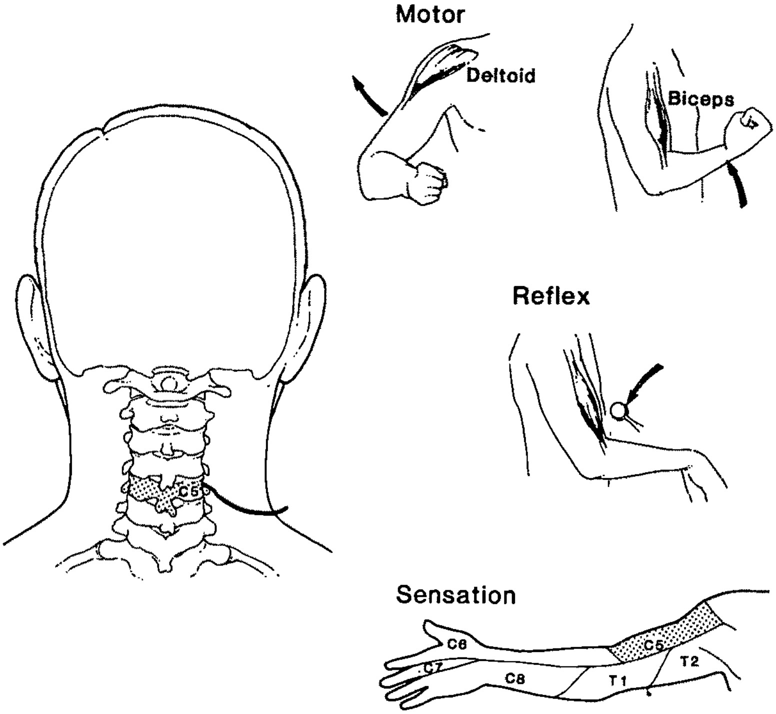

Figure 4. Sensory, motor, and reflex changes from C5 nerve lesions. (*From* Raj PP: *In* Raj PP (ed): Practical Management of Pain. Chicago, Year Book, 1986, p 432; with permission.)

ditionally, the primary service handled postoperative pain relief. Recently, however, anesthesiologists are becoming more active in this area. This may be because of (1) aging of the population, so that more patients with multisystem organ impairment, cardiovascular, and pulmonary pathologic conditions are being operated on; (2) increasing numbers of severely debilitated patients are being salvaged through modern intensive care unit management; (3) increasingly sophisticated and invasive surgical procedures with difficult pain management considerations are being performed; (4) increasing availability of patient-controlled analgesia, spinal opiates, and mixed agonist-antagonists (e.g., buprenorphine); and (5) recognition of pain management as a separate and evolving specialty by the medical profession, largely through the efforts of the American Academy of Pain Management.*

Systemic Analgesics

Systemic analgesics are the mainstay of therapy for acute pain. Narcotic analgesics are usually administered orally or intramuscularly, with ataractic/antihistamines (hydroxyzine, diphenhydramine), nonsteroidal anti-inflammatory drugs (ibuprofen), and benzodiazepines (lorazapam, diazepam, midazelam) used as adjuncts. Morphine sulfate is the most commonly used drug and is the standard against which other opioids are compared. Intramuscular use results in peak plasma levels in 20 minutes, 4 to 6 hours of analgesia, and a plasma half-life ($T_{1/2}$) of 2 to 4 hours. Oral doses are 3 to 6 times higher, because of hepatic metabolism (first pass effect), depending upon whether the patient is narcotive-naive.[55] Oral slow release tablets are available and have revolutionized the clinical practice of oncology.[25] Meperidine, which is 7.5 times weaker than morphine sulfate, provides 4 to 5 hours analgesia with a $T_{1/2}$ (orally) of 12 to 16 hours and a $T_{1/2}$ intramuscularly of 3 to 4 hours to normeperidine, a toxic metabolic that may produce central nervous system excitation[5] prior to renal excretion. Codeine, a good initial narcotic analgesic and antitussive, is 13 times weaker than morphine sulfate. It is administered orally in 200-mg doses. Duration of analgesia is 4 to 6 hours, with biotransformation to morphine sulfate in 3 hours.[25] Oxycodone is roughly equipotent with morphine sulfate. It is prescribed in 30-mg doses (orally) in combination with acetylsalicylic acid or acetominophen and lasts 3 to 5 hours. Hydromorphone is five times stronger than morphine sulfate; analgesia lasts 4 to 6 hours with a $T_{1/2}$ of 2 to 3 hours.[25] Methadone is equipotent with morphine sulfate and has good oral potency but requires careful titration to avoid accumulation, as the half-life is 18 to 30 hours.[27] Individualization of analgesic therapy is crucial to improve the quality of acute pain care. Many patients are undermedicated because of a misguided effort to reduce the risk of addiction (creation of addiction is rare in previously nonaddicted patients) or exaggerated fears of respiratory depression.[9, 22] Many patients are prescribed doses that are less than half of what is actually needed to relieve their pain.[9] Physicians have traditionally viewed their responsibility for pain control with an ambivalent concern for the patient's comfort balanced against fear of the potential adverse consequences of analgesics.[46] Analgesic requirements are cyclical and vary considerably among patients; however, they are consistent within each patient.[51] With careful titration they can be determined without the occurrence of complications.[61, 64, 68]

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Acute Pain Services (APS) have been established to provide analgesia for patients whose anticipated requirements are of a few days' duration. These in-hospital groups usually consist of an attending anesthesiologist, one or more anesthesiology/pain service rotating physicians, one or more nurse anesthetists, and a secretary. Other services, such as neurology, work with the APS on a part-time basis and supply referrals. The treatment modalities supplied by the APS are patient-controlled analgesia (PCA), spinal (epidural or intrathecal) opiates, anesthetic nerve blocks, and TENS.[49] APS treat postoperative patients, trauma patients, acute exacerbations or crises of chronic disease processes (sickle cell disease, pancreatitis), and oncology patients. A patient entering into the APS is referred by an attending physician. Written consent to any procedure must be obtained during

Table 2. *Stepladder Approach to Analgesic Tailoring*

Step 1	Aspirin-type drugs	Acetylsalicylic acid (ASA) Acetaminophen (Tylenol) Nonsteroidal antiinflammatory drugs
Step 2	Aspirin-type drugs and/or mild narcotics	ASA + codeine acetaminophen + codeine Propoxyphene (Darvon) Pentazocine (Talwin)
Step 3	Moderately potent narcotics	Oxycodone (Percodan) Hydrocodone (Vicodin)
Step 4	Highly potent narcotics	Meperidine (Demerol) Hydromorphone (Dilaudid) Dolophine (methadone) Levorphanol (Levodromoran) Oxymorphone (Numorphan) Heroin

From Ferrer-Brechner T: *In* Raj PP (ed): Practical Management of Pain. Chicago, Year Book, 1986, p 318; with permission.

therapy, daily progress notes are made, and the use of analgesics or sedatives is strictly limited to that prescribed by the APS. Coverage, preferably in-house, must be provided at all times.

Analgesics are titrated in a stepladder approach (Table 2). In addition to using the standard opioids, the APS also uses agents such as levorphanol, oxymorphone, or mixed agonist-antagonists, such as pentazocine, butorphanol, and buprenorphine. Use of these agents has been well-described.[22, 25, 53] Most patients are postoperative patients receiving PCA, which avoids the analgesic gaps of oral analgesics,[55] the peaks and valleys of intramuscular administration,[55] and the constant supervision and frequent titration necessitated by intravenous opioid infusion.[55, 64] PCA is an infusion device that allows the patient to self-administer small doses of narcotic through a button at the bedside.[64] The physician or nurse selects initial incremental doses (lockout), and total dose (4 hr maximum),[64] and an intermittent mode, continuous mode, or both is chosen. The device's microprocessor-controlled memory records the total number of patient attempts and the number of successful injections during the previous 1 to 12 hours to allow the operator to optimize the analgesic regimen.[64] The drug most commonly used for PCA is morphine sulfate (PCA dose, 5–30 mg; lockout, 5–15 minutes. Meperidine (PCA dose, 5–30 mg; lockout, 5–15 minutes) and hydromorphone (PCA dose, 0.1–0.5 mg; lockout, 5–15 minutes) are used for patients allergic to morphine sulfate.[1] Mixed agonist-antagonists are not good choices for PCA, as they have a ceiling effect on efficacy.[9, 14] Aside from surgical wards, PCA has applications in patients with sickle cell disease, in coronary care units, and in obstetric suites.[64]

Local Anesthetic Blocks

Many acute pain problems can be managed with local anesthetic blocks. For example, intercostal blocks can be used for rib fractures,[66]

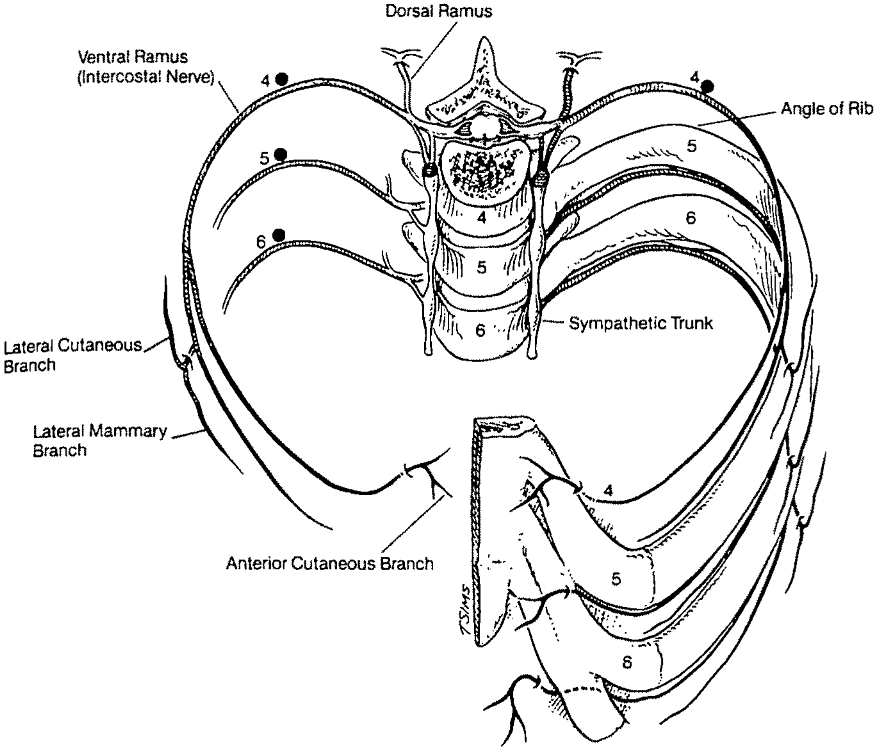

Figure 5. Anatomy of the intercostal nerve and its distribution with its branches along the ribs. (*From* Raj PP (ed): Handbook of Regional Anesthesia. New York, Churchill Livingstone, 1985; with permission.)

postoperative upper abdominal surgery, and thoracotomy pain.[14] Unilateral intercostal blocks significantly decrease postoperative opioid requirements after cholecystectomy and improve pulmonary function.[43] Intercostal blocks are performed at the inferior aspect of the desired rib dorsal to the lateral perforating branches on the posterior axillary line[45] (Fig. 5). Acute pain of the lower extremities, pelvis, abdomen, and thorax can be managed with intermittent or continuous local anesthetic blockade,[47] spinal (epidural or intrathecal) opiates,[18, 50, 67] mixed agonist-antagonists,[46, 67] intervertebral blocks,[46] or TENS.[54] Potential hazards with local anesthetic blockade include total spinal from intrathecal catheter migration, cumulative toxicity from systemic absorption, hypotension from sympathetic blockade, urinary retention, and excessive sensory or motor blockade.[17] Intraspinal opioids have been used in the treatment of acute and chronic pain.[17, 46] Morphine sulfate is the agent most commonly used epidurally (0.04 mg/kg + 0.015 mg/kg/h to a maximum of 0.1 mg/kg/day) and intrathecally (dose, 6–10 μg/kg/day). Long-acting, water-soluble narcotics (morphine sulfate) have a higher risk of respiratory depression than do short-acting, liposoluble agents (fentanyl, sufentanil).[1,

[67] Single-shot intrathecal fentanyl (6.5–10 μg) provides about 2 hours of analgesia; epidural fentanyl can be used as an infusion at 0.8 μg/kg/h. The doses listed are averages and may be altered as the clinical situation dictates. Epidural or intrathecal catheters may be left in situ for a continuum of perioperative analgesia, provided that rigid aseptic technique is maintained and the level of nursing supervision permits. Epidural buprenorphine can also be used postoperatively; a 100 μg bolus, followed by 30–60 μg/h infusion provides substantial analgesia with little respiratory depression.[67] Exclusion criteria for all of these techniques are patient refusal, inability to communicate with the patient, marked metabolic disorders (sepsis, severe fluid, or electrolyte abnormalities), a history of psychiatric disorders, and allergy to available agents. Relative contraindications are patients prone to respiratory depression (chronic obstructive pulmonary disease), end-stage renal disease, or hepatic disease. The risks of intraspinal opioids are higher in geriatric patients, at injections at more cephalad levels, and if concomittent sedatives or analgesics are adminsitered.[1, 46] All of the side effects are at least partially reversible with naloxone, with the exception that the bladder occasionally may need to be catheterized.[17] It is possible to reverse adverse effects without eliminating analgesia.[17] Nursing level of surveillance should be frequent and immediate availability of qualified personnel is essential if the patient becomes difficult to arouse or hypotensive or the respiratory rate drops below 12 beats/minute. Most studies show an extremely low incidence of clinically significant respiratory depression.[22]

Transcutaneous electrical nerve stimulation uses a battery-powered electrical pulse generator connected by a wire to two or more electrodes. Sterile, disposable, self-adhering electrodes are placed on either side of a wound at the time of tissue dressing application. The electrical current is increased until the patient feels strong paresthesias. TENS has limited application in postoperative and posttraumatic analgesia.[16] It decreases opioid requirements, improves lung function, decreases atelectasis and ileus.[3] Stimulation of inhibitory dorsal horn neurons through large afferent fiber activation is probably the mechanism of action.[39]

CHRONIC PAIN: THE PAIN CLINIC

Chronic pain is defined as pain that lasts beyond the time of healing or for more than 3 months.[40] It is frequently caused by degenerative processes; however a host of factors may be implicated, such as trauma, congenital deformities, inflammatory diseases, tumors, metabolic disturbances, neurogenic diseases, pathologic vascular and visceral conditions, emotional disturbances, and altered biomechanics.[2, 39] Many of these conditions cause various degrees of infirmity or remain partially treated, without ever being adequately investigated. Most of these patients do not need to be hospitalized.[39, 46] In this era of cost containment, there is emphasis on techniques that eliminate or shorten the hospital stay.[24] Chronic pain patients account for a disproportionate amount of resources and have poor results from therapy.[45] Many nonstandardized therapies have been involved in their management, such as acupuncture,[44, 46] chiropractics, massage, hypnosis and hydrotherapy.[46] An individualized, multidisciplinary approach is necessary in their management,[46] but this is often too time consuming for the primary physician.

The Pain Clinic is an organization wherein difficult pain patients are managed by a group of specialists in a clinic setting. These patients, with widely different diseases and clinical courses, are united in that the routine health care system has failed them. Many interesting iatrogenic and interdisciplinary problems are demonstrated so that the patient can be effectively managed with the combined knowledge and skills of anesthesiologists, neurosurgeons, psychologists, psychiatrists, orthopedic surgeons, neurologists, occupational, and physical therapists. Other services are called upon as needed. The Commission on Accreditation of Rehabilitation Facilities has established standards to be met when setting up a pain clinic (Table 3).[15] The ideal pain clinic is an independently budgeted, free standing business with a large referral population. It should be adjacent to, but autonomous from, a full-service medical center that extends admitting privileges of the pain clinic physicians to the hospital. Most pain clinics do not reach this level of complexity or autonomity.[46] The needs of a rural community may be met adequately with a much simpler design. Nationwide, most pain clinics offer only a few modalities: nerve block 87%, TENS 71%, physical therapy 62%, acupuncture

Table 3. *Services and Modalities for Outpatient Programs*

PRIMARY SERVICES	ADDITIONAL SERVICES
Clinical psychologist or psychiatrist Physical therapist Physician Specialized nurse	Alcoholism and drug-abuse treatment Chaplaincy Clinical laboratory Dentistry Diagnostic radiology Dietetics Exercise physiology Expressive therapies Medical/surgical subspecialty consultation Occupational therapy Orthotics Pharmacy Prosthetics Rahabilitation engineering Social services Therapeutic recreation Vocational services

From Standards Manual for Facilities Servicing People with Disabilities: Commission on Accreditation of Rehabilitation Facilities, 1989, p 53; with permission.

41.5%, psychotherapy 41%, behavior modification 38%, biofeedback 37.5%, two or more modalities 33%, drug detoxification 33%, hypnotherapy 27%, seven or more modalities 24%.[4] A simple, cost-effective pain service may be set up as a block clinic, an annex to a Department of Anesthesiology that offers limited procedures for acute and chronic pain problems.[1, 46] It is important to maintain flexibility and meet the changing needs of the patient population.

Sympathetically Maintained Pain

A major group of patients undergoing chronic pain therapy is comprised of those with sympathetically maintained pain (SMP). Causalgia is a syndrome of severe pain and autonomic dysfunction after a major nerve trunk injury.[17, 46] Reflex sympathetic dystrophy (RSD) is a syndrome of autonomic dysfunction and severe pain caused by microscopic nerve injury or illness (myocardial infarction).[17, 46] The frequency of RSD after peripheral nerve injury is 1% to 15%. Classic symptoms include burning pain, hyperesthesia, hard edema, hyperhidrosis, and trophic changes of the skin and bones.[17, 30] Pain and hyperalgesia (especially to light touch) often spread, not respecting dermatomal distribution.[46] The diagnosis is confirmed by diagnostic differential neural blockage: if pain can be eradicated by injection of a dilute concentration of local anesthetic (1% 2-CP chloroprocaine, 0.5% lidocaine) that leaves sensory perception intact, then it must be sympathetically mediated.[46] Many therapies have been used in the management of RSD.[10, 17, 30] Most commonly, the anesthesiologist performs a stellate ganglion local anesthetic blockade, trigger point injection, Bier blocks with bretylium (1.5 mg/kg) or guanethidine (30 mg in the upper extremity, 40 mg in the lower extremity), paravertebral nerve blocks, and TENS.[46] Guanethidine sulfate is an experiment drug and must be given according to the Ciba-Geigy protocol.[45]

Myofascial Pain

Another major group of referred patients have myofascial pain (MFP). In MFP, there is marked tenderness of discrete points within skeletal muscle (trigger points), pain referred to adjacent muscle sites, and tight, ropey bands of skeletal muscle.[46] Travell and Simons[1] suggest that the etiology of MFP is follows: acute muscle pain causes disruption of the sarcoplasmic reticulum and release of calcium, resulting in sustained muscle contraction. Adenosine triphosphate (ATP) becomes depleted, preventing release of actin from myosin and causing sarcomeres to become rigid. Scapulocostal syndrome (Fig. 6) is a common example. Treatment is aimed at gently increasing muscle length. Trigger point injections of local anesthetic agents (0.5% bupivacaine) are the mainstay of therapy; a session of physical therapy is ideally scheduled after each block. TENS and vapocoolant sprays are useful adjuncts.[18, 39]

Low Back Pain

A third major group of referred patients have low back pain and sciatica. The major causes of low back pain are lumbosacral radicu-

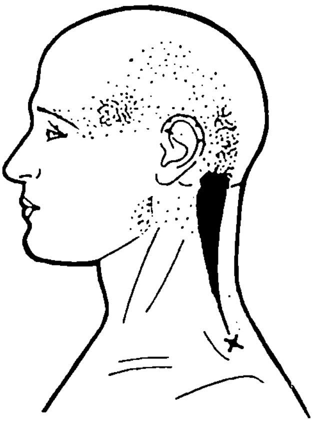

Figure 6. Distribution of myofascial pain affecting the trapezius muscle (*stippled area*) and trigger point (*X*). (*From* Ramamurthy T, Raj PP: Myofacial pain syndromes. *In* Raj PP (ed): Practical Management of Pain. Chicago, Year Book, 1986, p 424; with permission.)

lopathy (LSR), especially of the L5–S1 nerve roots (because of the narrow foramen) and lumbosacral arthropathy (LSA).[26] LSR and LSA are frequently difficult to differentiate because both conditions may result in back pain that radiates to the lower extremities[2] (Table 4). When the origin of low back pain is a single acute episode, 90% to 95% of patients recover spontaneously.[2] Sciatica occurs when there

Table 4. *Causes of Chronic Low Back Pain*

TYPE	CAUSE
Congenital	Spondylolysis, spondylolisthesis, facet asymmetry, transitional vertebra
Trauma	Ligamentous, muscle and joint strains, subluxations, dislocations and fractures
Degenerative	Osteoarthritis of spine, hip joints, spinal stenosis, disc degeneration
Inflammatory	Ankylosing spondylitis, arachnoiditis, cysts of dura, neuritis, rheumatoid arthritis
Tumor	
Benign	Neurofibroma, ependymoma, osteoid osteoma
Malignant	Multiple myeloma, metastatic, leukemia, osteogenic sarcoma
Metabolic	Osteoporosis, gout, diabetic neuropathy, Paget's disease
Endocrine	Hyperparathyroidism
Neurologic	Nerve root compression from any cause
Infection	Disc space infection, tuberculosis, epidural abscess, herpes zoster, meningitis, sacroiliac joint infection
Vascular	Ischemia of neural elements
Postoperative	Sequalae of scar formation
Emotional	Psychoneuroses, either primary or secondary
Scoliosis	Idiopathic, postparalytic, aging
Visceral	Visceral inflammation, female pelvic pathology, retroperitoneal pathology, aortic aneurysm, prostatic disease

From Addison RG: Review of chronic low back pain: Clin J Pain 1:50, 1985; with permission.

are mechanical and inflammatory stimuli, such as altered biomechanics, exposure to degenerating glycoproteins from a nucleus pulposus, and ischemic axonal degeneration from intermittent compression.[1, 2] Sciatica has a slightly more protracted course than acute low back pain but 50% of patients recover with 1 month.[26] Management of both conditions consists of diagnosis (back films, computed axial tomograms, bone scan), immobilization, rest, mild analgesics, and nonsteroidal anti-inflammatory drug administration (NSAID).[2, 17] Epidural steroid injections (methylprednisolone acetate 80 mg, triamcinolone diacetate 50 mg) with the affected side down often produces considerable relief.[1, 46] Facet arthropathies are also ameliorated by epidural steroid injections.[46] Simple treatment is advocated in most patients with low back pain and sciatica. Surgical intervention in a few patients with sciatica and neurologic claudication may become necessary; the diagnosis will usually be made by the neurologist, the neurosurgeon, or the orthopedist. However, long-term success after surgery for sciatica is poor (approximately 30%), and the results of long-term nonsurgical management are good (above 75%).[26] Patients with chronic low back pain or sciatica are poorer surgical candidates. If one spinal surgical procedure has been performed, there is a 30% chance that an additional procedure will improve pain.[2] If two or more spinal surgical procedures have been performed, the change of improvement decreases to 5%.[2] These patients are also less likely to benefit from epidural steroid injections, perhaps because of nerve scarring, inelasticity, and impaired steroid diffusion in fibrotic tissue.[1, 2] Aseptic meningitis from intrathecal injection occurs more frequently.[1] For resistant cases radiofrequency coagulation of the nerves supplying the facet joints,[46] cryoanalgesia,[46] rehabilitation and psychologic approaches have been advocated.[2]

Headache

Headache is a condition commonly treated at pain clinics and is the most frequent health complaint in the United States.[46] Its causes are many and varied and have been described elsewhere.[32, 33, 38, 68] Although it is most often treated by the neurologist, the anesthesiologist may be called upon to perform a greater occipital nerve block. This technique is used along with biofeedback in the treatment of occipital muscle tension headaches.[46] The greater occipital nerve is located halfway between the mastoid process and the greater occipital protruberance (inion) at the crest of the occipital bone; it can easily be blocked bilaterally at its origin with a minimum of complications.[46]

CONCLUSION

Inadequately treated pain syndromes represent a problem of enormous magnitude. Aside from the waste of human resources, there is an untold loss in the quality of life of a large segment of the US population. The emotional and psychologic suffering of patients and their families is an additional factor. Even with optimal medical care, most patients never reach their full functional capacity unless the issue of pain control is addressed adequately.

To enter the pain management field a basic knowledge of neuroanatomy together with certain clinical skills is required. A block clinic may be easily formed with referrals from neurology, orthopedic surgery, and neurosurgery. Intraspinal opiates may be administered to patients in the intensive care units, along with epidural steroids, intercostal blocks, and TENS. If feasible, the service may be expanded to include PCA, and various supportive care services, such as physical therapy, orthotics, rehabilitation medicine, psychotherapy, and detoxification therapy. A liaison formed with a neurosurgeon for the evaluation and placement of permanent epidural or intrathecal opioid infusion devices, coupled with visits from a reliable home health care service, may prove especially rewarding in the management of outpatients with certain end-stage neoplasias. For anesthesiologists to be successful, they must be flexible to meet the changing needs of their community and must know where to refer patients who will benefit from services that they do not offer.

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Preanesthetic Assessment of the Oncology Patient

*Richard B. Patt, MD**

After cardiovascular disease, cancer ranks as our nation's number one cause of mortality, as it accounts for an estimated 510,000 deaths annually in the United States.[22] Despite an increased emphasis on early diagnosis, improvements in the cure rate for cancer continue to increase only incrementally (global 5-year survival of 40%),[5] but methods of palliating the disease and extending life have progressed, resulting in greater numbers of patients with advanced cancer who present for surgery and anesthesia.

Cancer refers generically to a multitude of different disorders that share certain histopathologic features but that vary considerably in their potential to invade locally, spread to distant sites, and to interfere with physiologic function and ultimately shorten life. Although a heterogenous disease, cancer frequently is characterized by involvement and dysfunction of multiple organ systems. On this basis alone, the potential for difficulty in providing safe anesthetic care and protection from surgical stress increases in patients with advanced or highly malignant disease. In addition, cancer therapy is responsible for a variety of biologic injuries that have the potential to impair normal physiologic function further and that add to the risks of anesthesia and surgery. Finally, patients are often multisymptomatic. The symptoms of cancer as well as the effects of ongoing palliative treatment must be assessed for potential impact on perioperative care.

SURGERY

As stated by Howland and colleagues,[13] preoperative evaluation of the extent and nature of oncologic disease is complemented

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by the intraoperative surgical assessment. Planned major surgery may be aborted when disease that is initially considered curable is determined at the time of surgery to have metastasized, thus making resection unwise. Conversely, surgery may be unexpectedly prolonged when intraoperative findings suggest that planned palliative surgery demands a curative approach. Anesthesiologists must be prepared for altered surgical approaches and anesthetic requirements.

PARENCHYMATOUS, LOCAL, AND INDIRECT EFFECTS

Virtually all organ systems are subject to failure based on malignant infiltration or external compression by tumor. The significance of local effects vary according to location and tumor bulk. Perioperative management of organ failure is similar in most respects, whether the cause is oncologic or nononcologic, so only features specifically related to oncologic disease are highlighted herein.

Primary or metastatic invasion of the liver, kidneys, bone marrow, lung, and other organs may be present with varying degrees of organ dysfunction. A thorough history, physical assessment, and review of laboratory findings is essential to determine the presence of organ failure so that anesthetic management can be altered accordingly.

Pathologic states resulting from external compression of vital structures often progress chronically and insidiously. Adequately compensated in the awake patient, they may go unrecognized until uncovered by either the stress of anesthesia and surgery or a careful preoperative assessment. Problems with the potential to influence normal responses to anesthesia include obstruction of the airways, superior vena cava, or gastrointestinal tract, pericardial tamponade, raised intracranial pressure, spinal cord compression, pancreaticobiliary obstruction, and renal failure.

Finally, a variety of metabolic, nutritional, psychologic and hematologic disorders may result indirectly from various humoral aspects of malignant disease.

HEMATOLOGIC EFFECTS

Anemia

Anemia is common and often multifactorial; it arises from several causes including occult or acute hemorrhage, tumor infiltration of bone marrow, nutritional deficiencies, hemolysis, hypersplenism, and chemotherapy or radiotherapy. Standard preoperative investigations including a careful physical examination and complete blood count will detect anemia and other hematologic abnormalities.

Anemia is often chronic and well-tolerated, particularly in the setting of a sedentary lifestyle. An individualized approach to preoperative transfusion is now recommended in preference to uniform repletion to a hematocrit level of 30%.[9] When contemplating transfusion, important considerations include dysfunction of other end organs, particularly of abnormalities that might influence their demand for oxygen, and the anticipated stress and blood loss associated with the planned surgical procedure.

Coagulopathy

Depletion of coagulants and even disseminated intravascular coagulation (DIC) may result from many of the same factors that predispose one to anemia. Additionally, patients who have recently had chemotherapy may be predisposed to DIC and hemorrhage. Many types of tumor cells (promyelocytic leukemia, adenocarcinoma of the prostate) are rich in thromboplastin-like materials. Necrosis induced by cytotoxic drug therapy can result in massive release of this material.[1] Subsequent consumption of fibrinogen and other clotting factors can lead to DIC and hemorrhage.

As with most disorders of coagulation, a careful patient history and physical examination frequently will uncover potential problems. Screening with measurements of prothrombin time, partial thromboplastin time, platelets, and bleeding time is further recommended in patients with advanced or metastatic carcinoma and after recent chemotherapy or radiotherapy. In the presence of abnormalities of these tests, a more exact diagnosis is often suggested by analysis of levels of fibrinogen and fibrin split products as well as with an examination of a peripheral blood smear that, after chemotherapy, may reveal abnormal red cells and red cell fragments.

Thrombocytopenia may result from these causes. Bone marrow suppression produced by hemibody radiation and many chemotherapeutic regimens usually results in the lowest peripheral counts (nadir) 8 to 10 days after treatment, although there is interagent variation. Patients with platelet counts below 30,000 are at risk for spontaneous hemorrhage, thus when counts are in this range and major surgery is planned, authorities recommend preoperative platelet transfusion.[23]

Preoperative restoration of normocoagulation is essential in the patient with a bleeding abnormality. Depending on the severity and cause of the underlying bleeding disorder as well as the urgency of surgery, therapy may include treatment with vitamin K, fresh frozen plasma, whole blood, platelets, or heparin.

Polycythemia

In polycythemia vera, a myeloproliferative disorder, hematocrit levels often exceed 55%, and in about one half of cases platelet counts are greater than 500,000. In addition, hypernephroma and hepatoma are occasionally associated with an increased production of erythropoietin and, consequently, an increased red cell mass. Hyperviscosity of blood and stasis predispose the patient to vascular thrombosis and platelet defects, which may contribute to hemorrhage. A phlebotomy to reduce the hematocrit to near normal levels is recommended before elective surgery. Dilution with infused solutions of crystalloid is indicated in emergency situations to reduce the incidence of thromboembolic phenomena.

Leukostasis

This phenomena is seen almost exclusively in patients with leukemia and high circulating volumes of blast cells. Leukemic cells plug small capillaries, resulting in multiple microinfarctions and hemorrhage and often producing prominent pulmonary and central nervous system symptoms. If this syndrome goes unrecognized it can be fatal.[1] The diagnosis is determined by patient history, physical examination, and analysis of a peripheral blood smear. In the presence of leukostasis, surgery should be postponed until urgent chemotherapy or leukopheresis can be undertaken.

METABOLIC EFFECTS

Fever

Immune system function is often abnormal in patients with cancer and predisposes them to infection and temperature elevations. A source of the fever should always be sought so that specific treatment can be instituted, but pyrexia in the absence of demonstrable infection is relatively common in patients with advanced cancer. So-called neoplastic fever is particularly common in the presence of liver metastases and hematologic malignancies, especially Hodgkin's disease.[8] Proposed mechanisms for neoplastic fever include tumor necrosis, peritumoral inflammation, and tumor-mediated release of endogenous pyrogens. Affected patients may be prescribed naproxen or corticosteroids, which have been demonstrated to induce fever lysis in patients with neoplastic fever.

Lactic Acidosis

Lactic acidosis with consequent tissue hypoxia can result from a variety of causes, but it is usually related only indirectly to tumor growth. Mortality is high and vigorous efforts should be made to correct abnormalities before considering all but the most urgent surgery. Infrequently the highly proliferative malignant processes such as acute leukemia and lymphoma can result in lactic acidosis, probably as a consequence of massive local anaerobic glycolysis. In cases of tumor-induced lactic acidosis, extensive neoplastic invasion of the liver is usually present.[17] Symptomatic treatment is warranted until long-term resolution can be obtained by treatment of the underlying disease with antitumor therapy.

Hypercalcemia

Hypercalcemia occurs when entry of calcium into the extracellular fluid, usually from bone resorption, exceeds the kidneys' ability to clear calcium. Metastatic invasion of bone is the most common cause of hypercalcemia in patients with cancer; most frequently the initial cancer is a primary tumor of the breast or lung, multiple myeloma, or renal cell carcinoma. In addition, serum calcium levels occasionally are elevated by the ectopic production of parathormone by tumors involving the kidney, lung, ovary, or pancreas or by tumors that produce large quantities of prostaglandins. About 10% to 20% of patients with cancer will develop hypercalcemia at some stage of their disease.[11]

Normal levels of serum calcium range from 9.5 to 10.5 mg/dL. Elevations to levels of up to 12 mg/dL are often well-tolerated, but calcium levels of 14 mg/dL produce symptoms in most patients. These symptoms of mild hypercalcemia include nausea, vomiting, constipation, and urinary frequency. As serum calcium rises, progressive lethargy and confusion can progress to coma and death, which is usually secondary to disturbances of cardiac rhythm. Characteristic alterations in cardiac conduction include prolongation of the P-R interval, widening of the QRS complex, and shortening of the Q-T interval. Hypercalcemia is exacerbated by dehydration and immobilization.

Severe (14–16 mg/dL) or markedly symptomatic hypercalcemia requires urgent treatment. Initially, vigorous intravenous hydration with normal saline is used to dilute serum calcium and increase its renal loss. Furosemide is usually administered to reduce the risks of overhydration and to enhance calciuria further. Monitoring of cardiac filling pressures is recommended. Digitalis toxicity as well as hypokalemia may occur, and potassium replacement is recommended. Corticosteroids are frequently administered to increase urinary losses of calcium further. Mithramycin, a cytotoxic antibiotic, inhibits bone resorption and effectively lowers serum calcium in a high percentage of cases, usually within 24 to 48 hours of administration. Once hypercalcemia has been controlled, chemotherapy or radiotherapy should be planned to prevent recurrence.

PARANEOPLASTIC SYNDROMES

Squamous cell tumors of the lung and renal cell carcinoma account for more than 50% of cases of ectopic production of parathormone.[15] Tumor production of prostaglandins also has been implicated as a cause of hypercalcemia and suggests the use of indomethacin to inhibit prostaglandin synthesis.[21] Recently, osteoclast activating factor, a peptide elaborated by certain tumor cells, has been identified as causative factor of hypercalcemia in some patients. Glucocorticoids have been observed to inhibit the activation of osteoclasts by osteoclast activating factor.[11]

Other paraneoplastic syndromes have been identified and, rarely, may be responsible for Cushing's syndrome, the syndrome of the inappropriate secretion of antidiuretic hormone, thyrotoxicosis, and hypoglycemia. A variety of multiple endocrine adenomatoses also have been identified and should be reviewed for potential impact on anesthetic management.[17]

Of particular interest to the anesthesiologist is the Eaton-Lambert or myasthenic syndrome, which is observed most often in association with carcinoma of the bronchus but that also may accompany thymoma and other tumors. Affected patients are extremely sensitive to both nondepolarizing and depolarizing muscle relaxants, their response to anticholinesterase agents is often poor, and the need for postoperative ventilatory support is common.[10]

NERVOUS SYSTEM

Spinal Cord Compression

Compression of the spinal cord by neoplasm, when amenable to operative intervention, represents a true surgical emergency. In a series of 150 patients with metastatic spinal cord compression, urgent surgery resulted in relief of pain in 75%, ambulation in 66%, significant return of motor function in 16%, and maintenance of ambulation and continence in 43% 6 months after surgery.[24] Unfortunately, the onset of progressive neurologic symptoms is often insidious and may go unrecognized, particularly in patients already multisymptomatic, until late signs are already established. The presence of dermatomal sensory loss and frank muscle and bladder paresis usually precede irreversible paraplegia or quadriplegia by only 12 to 24 hours and when present necessitate prompt diagnosis and therapy. If spinal cord compression is suspected, urgent radiologic evaluation by myelography or magnetic resonance imaging is undertaken. Once a diagnosis has been established, high dose corticosteroids are administered and emergency decompressive laminectomy is considered. Factors favoring surgical intervention include rapid progression of symptoms, absence of a histologic diagnosis, and poor radiosensitivity. Postoperative radiation is now commonly used. As an alternative to surgery, emergency high-dose radiotherapy is considered in the presence of highly radiosensitive tumors, multiple or extensive lesions, a slow progression of symptoms, and when patients are not otherwise fit for surgery.

Before the operation, patients and family members may be anxious, as they contemplate the prospect of paralysis, major surgery, and further disability. A thorough, compassionate explanation of anesthesia and, when the medical condition is sufficiently stable, generous premedication are indicated. Radiologic findings are reviewed and discussed with the surgeon to determine if any instability of the cervical spine is present that may require consideration of special airway management such as fiberoptic, nasal, or conscious intubation to minimize further neurologic injury. If conscious intubation is elected, topical anesthesia and sedation should be used to reduce coughing.

Immobility may predispose the patient to pulmonary embolism and atelectasis. Dehydration may be present as a consequence of chronic illness and steroid administration. In patients with advanced lesions, sympathetic nervous system dysfunction may result in impaired cardiovascular compensation for alterations in posture and blood loss. Preoperative hydration with a balanced salt solution and prompt replacement of blood loss are indicated to attenuate intraoperative reductions in blood pressure. Although intraoperative blood loss for elective spinal stabilization is not generally excessive, the mean loss in a group of 117 patients operated on for metastatic disease was 2137 ± 1566 mL, which doubled in a smaller group of patients when an anterior approach was required.[24] Measurements of cardiac filling pressures are a useful guide to fluid therapy. Patients may become poikilothermic below the level of injury, and normothermia should be aggressively maintained.

Patients with high lesions should be assessed for weakness of muscles of respiration and should be observed carefully once sedatives have been administered. The risk of succinylcholine-induced hyperkalemia increases during the first few days after neurologic injury. Because the time of initial neurologic insult is often indeterminable a nondepolarizing agent should be selected when possible to facilitate tracheal intubation. Hypertension and bradycardia associated with autonomic hyperreflexia are only of concern when denervation is chronic.

BRAIN METASTASES

As with primary tumors, brain metastases are often associated with pathologic elevations of intracranial pressure. Symptoms often respond to conservative therapy including fluid restriction, high-dose corticosteroids, osmotic diuretics, anticonvulsants, and total brain irradiation. Patients with meningeal involvement are candidates for intrathecal chemotherapy and may present for monitored anesthetic care for placement of an Ommaya reservoir. If hydrocephalus is present as a consequence of obstructed cerebrospinal fluid circulation, needle drainage by a burr hole made under local or general anesthesia or placement of a ventriculoperitoneal shunt may be palliative. Surgical resection or decompression of metastases is attempted occasionally particularly if the cerebral lesion is solitary or the primary neoplasm has not been identified.

Patients with brain metastases may require unrelated palliative surgery. Because the patient may be confused, informed consent must be obtained from a legal guardian. Premedication should be adminsitered only under direct supervision and with careful monitoring, as the patient's confusion may obscure early signs of hypoxemia. Premedication should be omitted altogether in the presence of elevated intracranial pressure to avoid hypoventilation and further elevations in intracranial pressure secondary to hypercarbia-induced increased cerebral blood flow.

RENAL ABNORMALITIES

Hyperuricemia is associated mainly with lymphoproliferative and myeloproliferative disorders and is most often the result of aggressive chemotherapy or radiotherapy. The destruction of large numbers of malignant cells releases large quantities of nucleic acids and leads to accumulation of uric acid, which is the major product of purine catabolism. Precipitation of uric acid crystals is associated with gouty arthritis and varying degrees of renal injury, the most important of which is acute hyperuricemic nephropathy. Sudden onset of oliguric or anuric renal failure is characteristic and is caused by obstruction at the level of the distal nephron. Clumping of crystals within the ureters may occur and is associated with flank pain. Hyperuricemia is a common secondary feature of renal failure, regardless of its cause, but crystalluria and a disproportionate elevation in serum uric acid levels in susceptible patients suggest that it is causative.

Prior to cytotoxic therapy, prophylaxis should be started in at-risk patients. Treatment of established acute renal failure involves vigorous intravenous hydration, careful alkalinization of the urine with intravenously administered sodium bicarbonate, and the administration of allopurinol, an inhibitor of the enzyme xanthine oxidase. Mannitol has been used successfully to reestablish urinary flow, and in otherwise unresponsive cases, treatment with dialysis is indicated.

ADRENAL GLAND

Hyperadrenocorticism (Cushing's syndrome) may be related to increased levels of adrenocorticotropic hormone (ACTH) from pituitary abnormalities (30% of patients), primary malignancy or adenoma of the adrenal cortex (30%), the exogenous administration of steroids, and more commonly than previously appreciated, ectopic production of ACTH (30%).[18]

The manifestations of Cushing's syndrome are protean and include hypertension, hypernatremia, hypokalemia, hyperglycemia, hypervolemia, skeletal msucle weakness, osteoporosis, and psychologic disturbances. In its chronic form, physical manifestations include central obesity with wasting of the distal limbs, cutaneous striae, accumulation of fat in the face (moon face) and between the scapulae (buffalo hump).

Electrolyte abnormalities should be corrected before surgery. If potassium repletion is necessary, it should, when possible, occur during days or weeks to replenish total body potassium stores. Patients should be assessed preoperatively for signs of congestive failure, and diuretic therapy should be considered.

Careful positioning of the patient during surgery is important to minimize trauma to fragile integument and to avoid fracture and undue pressure on spinal nerve roots from bulging discs and bone spurs.

Hypothalamic-pituitary-adrenal axis suppression, with the potential for impaired intraoperative stress response (Addisonian crisis) should be suspected in patients who have been exposed to steroids either chronically or for a short time. Accordingly, it is common to infuse the equivalent of 300 mg intravenous hydrocortisone in the perioperative period, usually in divided doses.

SUPERIOR VENA CAVAL SYNDROME

Superior vena caval syndrome has been observed in 3% to 8% of patients with bronchogenic carcinoma and is less commonly associated with malignant lymphoma, mediastinal fibrosis, and substernal thyroid.[7] Physical findings include engorgement of the superficial veins of the head and neck, brachiocephalic edema, and cyanosis. Orthopnea related to compression of the pulmonary vessels is common, and postoperative patients should remain in the semirecumbent position. Because conjunctival edema may be present, care should be taken to protect and lubricate the eyes while under anesthesia. The initial treatment is with radiation to reduce mediastinal tumor bulk. Mucous membrane engorgement may complicate airway management, and if surgery and anesthesia cannot be postponed, awake fiberoptic intubation should be considered along with surgical support for emergency cricothyrotomy.

PERICARDIAL EFFUSION/TAMPONADE

Malignant pericardial effusion is most often the result of direct spread of disease from mediastinal lymphomas and bronchogenic or esophageal carcinomas.[19] As with nonmalignant effusions, symptoms reflect the rate of fluid accumulation and its absolute volume. Pulsus alterans and paroxysmal atrial tachycardia, fibrillation, or flutter may be observed. Diagnosis of pericardial effusion is confirmed with echocardiography.

Pericardial tamponade results when accumulated fluid produces elevations in intracardiac pressures and the subsequent reductions in diastolic filling, ejection fraction, and blood pressure. Compensatory tachycardia, vasoconstriction, and increased venous return sustain cardiac output as long as central venous pressure exceeds right ventricular end-diastolic pressure.

CHEMOTHERAPY

Most chemotherapeutic drugs act by interfering with the synthesis or function of DNA or RNA in highly proliferative cells. A certain degree of toxicity among normal cell populations is inevitable in the course of administering chemotherapy. Toxicity is most often manifest by side effects on highly proliferative nonmalignant cells, notably those of the bone marrow, gastrointestinal mucosa, skin, hair follicles, and gonads. Side effects include anemia, thrombocytopenia, agranulocytosis, nausea, vomiting, dermatitis, and hair loss. In addition, toxicity to other organ systems may occur and influence the conduct of anesthesia.

Pulmonary Fibrosis

Pulmonary fibrosis is most closely associated with the administration of bleomycin, an antibiotic used to treat testicular and other cancers, and less often with busulfan, carmustine, and mitomycin-C therapy. A reduced diffusion capacity and total lung capacity is present in one third to one half of patients exposed to bleomycin, although fibrosis is clinically significant in only about 5% of treated patients.[2] In cases of significant fibrosis, initial symptoms such as dyspnea and nonproductive cough are insidious in onset and are typically followed by bibasilar rales and radiographic changes, that is, findings indicative of irreversible disease. Patients with advanced age or preexisting pulmonary disease appear to be more susceptible to the development of pulmonary fibrosis.

High incidences of postoperative adult respiratory distress syndrome and pulmonary mortality have been observed in bleomycin-treated patients. The recognition of similarities between pathologic specimens in bleomycin-treated patients who died postoperatively and those of patients with oxygen toxicity motivated a prospective study by Goldiner and colleagues[12] in which intraoperative fraction of inspired oxygen (F_{IO_2}) was limited to 0.22 to 0.25 and in which there were excellent results. Although the conclusions of this study have been challenged,[14] most authorities recommend using a reduced F_{IO_2} in susceptible patients. Limiting the volume of fluid administration and administering colloids in preference to crystalloids has also been recommended to reduce the risk of postoperative pulmonary edema.[12]

Cardiac Toxicity

Cardiac toxicity is associated with the anthracycline antibiotics, doxorubicin and daunorubicin. Analysis of several studies suggests that toxicity is minor in about 10% of patients and usually consists of transient electrocardiogram abnormalities but that about 2% of patients experience major toxicity with cardiomyopathy and heart failure. In affected patients, the syndrome was irreversible in 59% and was followed by death within about 3 weeks.[20] The risk of cardiomyopathy appears to be increased in patients with pre-existing heart disease and after mediastinal radiation.

In the minor variant, electrocardiographic changes are nonspecific, varied, and do not appear to reflect underlying cardiomyopathy. Changes observed include ST segment and T wave abnormalities, reduced QRS voltage, supraventricular tachycardia, premature atrial and ventricular beats, conduction abnormalities, and left axis deviation. In the case of major toxicity, cardiomyopathy is usually preceded by a dry, nonproductive cough and rapid progression of heart failure that is characteristically poorly responsive to pharmacologic and mechanical intervention.

RADIOTHERAPY

Injury to the structure and function of normal surrounding tissue is an accepted and common sequel to radiotherapy. The suppressive effects of radiation on bone marrow already have been noted. Radiation pneumonitis occurs most frequently during the course of radiotherapy but also may arise months after treatment. The acute endothelial injury and increased vascular permeability may result in edema and hemorrhage, but the chronic condition is one of restrictive disease. Postirradiation pericarditis may occur. Gastrointestinal and respiratory mucosa are highly radiosensitive. Airway manipulation may produce hemorrhage or edema in early phases that results from hyperemia and tissue fragility; chronically, fibrotic strictures may complicate intubation. Likewise, within the first 2 weeks after radiation exposure, skin may be fragile and erythematous, thus reducing intravenous access and requiring protection from trauma intraoperatively. Subsequently, chronic skin changes of atrophy and fibrosis may limit intravenous access.

CANCER SYMPTOMS AND PALLIATIVE CARE

Patients with cancer tend to be multisymptomatic; symptoms usually increase in number and severity as disease progresses. Pain is the most common symptom overall, and is a significant problem in two thirds of patients.[3] Treatment with oral opioid analgesics with or without nonsteroidal antiinflammatory agents and adjuvant drugs (antidepressants, anticonvulsants, amphetamines) relieve pain effectively in most patients, and is an accepted standard.[6] Patients may be taking high doses of opioids by a variety of routes (oral, subcutaneous, intravenous, epidural). When taken orally, morphine is often administered in slow-release preparations for basal analgesia, supplemented by immediate release preparations for breakthrough pain. Tables describing equianalgesic doses and pharmacodynamic and pharmacokinetics profiles for commonly used analgesics are available and are a useful reference for perioperative management.[16]

Maintenance of a high quality of life and a pain-free existence are acknowledged as important goals in the management of cancer patients, thus chronic opioid use is medically legitimate and should proceed uninterrupted. Additionally, physical dependence is common and narcotic administration should be continued to avoid the emergence of an abstinence or withdrawal syndrome. Likewise, the perioperative use of narcotic antagonists should be avoided. A potent volatile anesthetic is usually an important component of anesthesia, as patients tend to be tolerant to the effects of narcotics and many are already consuming doses sufficient to produce anesthesia in narcotic-naive subjects.

Other symptoms and their treatment have potential impact on anesthetic management and should be evaluated. Gastrointestinal dysmotility is common because of autonomic dysfunction (particularly after vincristine), opioid-induced constipation, circulating humoral factors, and mechanical obstruction. The presence of nausea, vomiting, or early satiety suggest the need for awake or rapid-sequence endotracheal intubation. Patients are frequently prescribed antiemetics (as well as anxiolytics and antidepressants) that may contribute to delayed emergence from anesthesia. In addition to their other multiple uses, steroids (as well as megestrol, a progestational agent) may be prescribed to enhance appetite and mood, thus indicating perioperative steroid replacement. Recently, central nervous system stimulants such as dextroamphetamine and methylphenidate have been recommended to decrease opioid-mediate drowsiness.[4] These drugs act to release stored norepinephrine. Anesthetic requirements may be increased with their short-term use or reduced with chronic use; in either case increased arrhythmogenicity is possible.

SUMMARY

The perioperative management of patients with cancer is challenging for anesthesiologists for many reasons. Proposed procedures range from radical, potentially curative operations that tend to be lengthy and involve large amounts of blood loss to relatively innocuous palliative interventions in cachectic patients who have multiple organ failure. Surgical plans are frequently altered in midstream. Patients may suffer from a variety of physiologic dysfunctions, either obvious or occult, only some of which can be detected preoperatively. Many patients have been exposed to noxious antitumor therapy that resulted in damage to tissue uninvolved by tumor. Patients are often multisymptomatic and as a result are exposed to a variety of medications that may interact with their anesthetic regimen. Finally, ethical issues of life support and resuscitation need to be addressed with the surgeon, patient, and family members before proposed surgery.

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Preanesthetic Assessment of the Patient with Acquired Immunodeficiency Syndrome

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HISTORICAL BACKGROUND

In 1968 a sexually active 15-year-old boy was admitted to the St. Louis City Hospital with an extensive chlamydia infection and a small lesion on his thigh. He did not respond to treatment and unfortunately, and unexpectedly, died. The postmortem examination of the small thigh lesion revealed that it was an unimpressive exterior manifestation of an intra-abdominal, aggressive, parenchymatous Kaposi's sarcoma. In 1988, serum and tissue specimens that had been frozen and preserved at the time of autopsy were re-examined, and the presence of human immunodeficiency virus (HIV) type 1 was found. The boy was thus the first identified, albeit retrospectively, patient with the acquired immunodeficiency syndrome (AIDS) in the United States.[12] In 1968 the disease was unknown, and the patient's illness an unexplained medical mystery.

In 1981 there appeared two concurrent reports of a previously undescribed syndrome. In California, four previously healthy homosexual men developed *Pneumocystis carinii* pneumonia, extensive mucosal candidiasis, and multiple viral infections.[13] One patient developed Kaposi's sarcoma, and cytomegalovirus was recovered from all four. All the patients were anergic, lymphopenic, and immunodeficient. The authors suggested that the syndrome was caused by the cytomegalovirus infection. In New York,[16] 11 cases of community acquired *P. carinii* pneumonia, occurring between 1979 and 1981, were investigated. Young men who were drug abusers, homosexual, or both, presented with pneumonia and lymphopenia. One patient had Kaposi's sarcoma, and 8 of the 11 were already dead at the time of the report. The authors suggested that drug abusers and homosexual men may be at high risk for pneumocystis infection.[16]

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The cause of AIDS is a retrovirus, the HIV.[3] At least two distinct variants of HIV have been identifed, HIV-1 and HIV-2.[4] Each variant can cause AIDS, but HIV-1 is known to have caused most of the AIDS cases in Africa, Europe, North America, and South America. It has been suggested that AIDS induced by HIV-2 may be milder and not as readily transferred from mother to child as is HIV-1-induced AIDS. Proof is lacking, however, as few HIV-2-induced cases of AIDS have been identified, and the incubation period from time of infection to obvious manifestation of the disease if often very long. Because screening tests for HIV-1 often do not detect HIV-2, the actual number of HIV-2-infected people in the United States is unknown. Each virus, HIV-1 and HIV-2, has been identified in Africa, Europe, and the United States, each is known to have caused AIDS, and each is a contaminant of the American blood supply.

EPIDEMIOLOGY

AIDS probably developed in Central Africa, perhaps as a particularly malignant mutant of the human T-cell lymphotropic virus (HTLV) family identified in both humans and monkeys. Seroprevalence rates in sub-Sahara Africa range from almost undetectable in some areas to as high as 18% in others. It is estimated that as many as 50 million Africans will be infected with HIV by 1993, that 1 million will be dead by 1991, and another 10 million dead between 1992 and 1996.[24]

Morbidity and mortality from AIDS is a serious public health problem in the United States and will continue to increase in the foreseeable future. The effects will be especially devastating in the young and middle-aged population of black and Hispanic men. By the end of 1989 117,781 cases of AIDS had been reported in the United States; there had been 70,313 deaths.[7]

There is a definite geographic prevalence of the disease in the United States, although it has been reported in each of the 50 states: 22.8% of all cases have occurred in New York, 20.3% in California, 8.7% in Florida, 7.1% in Texas, and 6.9% in New Jersey.[7] The New York State Commissioner of Health estimates that by the end of 1988 there were 20,000 cases of AIDS in New York and 10,000 deaths. By 1994, there will be 90,500 cases and 20,000 deaths. The actual number of HIV-infected people in the United States (most of whom have no knowledge of their infected state) is unknown, but estimates range from 800,000 to 1.5 million. Thus, it is certain that the practicing anesthesiologist will deal with HIV-infected patients: those who are infected, asymptomatic, unrecognized, and *in extremis*.

AIDS: THE DISEASE

To evaluate the AIDS patient, the anesthesiologist must have some knowledge of the nature of the disease, its signs and symptoms, the risk to health-care workers who participate in the treatment of these patients, and the risk the health-care worker represents to his patients.

AIDS is an attack upon the cell-mediated immune system, which may be considered a system of checks and balances, with T4 (helper) cells of the lymphocyte series acting as guardians and recognizing foreign invaders, whether viral, bacterial, or protozoan. When the body's integrity is disrupted the system has the capacity to recognize the disruption and to mobilize the immune system by humoral transmitters. The host defense reaction is discontinued by T8 (suppressor) cells. HIV invades and inactivates T4 cells while having no effect on T8 cells. The cell mediated immune system is, in effect, turned off, allowing the development of opportunistic infection by organisms ordinarily held in check by the intact and functioning immune system.[25] At the same time, a wide variety of tumors are allowed to develop and grow, uninhibited by the natural defense mechanisms.

Laboratory tests for the virus antigen are available, but they are expensive and unreliable. Thus, it is usual to search for anti-HIV antibodies as an indicator of infection. The enzyme-linked immunosorbent assay (ELISA) is most commonly used because of its low cost, simplicity, and capacity to be performed readily on a large scale. Western blot analysis or immunoprecipitation assay (RIPA) may be used to confirm a positive ELISA. Although it has been suggested that large segments of the population and indeed, perhaps all residents of the country, should be tested for anti-HIV antibodies, such suggestions are both simplistic and impractical. After infection with HIV the ELISA usually remains negative for 8 to 12 weeks. The more sensitive RIPA will become positive in 2 to 3 weeks. However, viremia will develop in as little as 8 days.[11] A "window in time" thus exists during which the contaminated individual will be capable of infecting others but will still register as uninfected by the usual screening and confirmatory tests. In some instances seronegativity for antibodies may persist for as long as 14 months in infected individuals who have the ability to infect others.[20]

The value of extensive screening is decreased even more by the high rate of false-positivity that occurs when screening certain population groups. For example, if the tests are used to screen populations with a high rate of true prevalence of HIV, then the false-positive rate is low. If, however, a population is tested in which the true rate of positivity is low, then the rate of false-positives may exceed 50%.[17] Although it may be important to identify infected individuals, it is at least as important not to label someone as HIV-positive when, in fact, they are not infected. The stigma associated with the disease and the promise of a lingering and painful illness and death make it imperative that no false diagnoses of HIV positivity be made. The Armed Services has developed a refined system of testing and retesting for HIV positivity and has reduced the false-positive results to 1 in 135,187 tests.[5] Yet the cost of the technique (estimated at $40,000 per person tested) makes its widespread use impractical.[29] The value of widespread screening is further vitiated because screening for HIV-1 does not reliably screen for HIV-2. Thus, presence of HIV-2 infection can only be detected with screening devices aimed specifically at HIV-2.

Methods of Transmission

In contrast to other communicable diseases, most notably hepatitis B, it is remarkably difficult to transmit HIV from an infected person to one not infected. Although the virus has been identified in seminal fluid, blood, tears, saliva, and synovial fluid and, indeed, no body fluid can be thought of as completely safe, the most important vector of transmission is blood. Even though AIDS is a sexually transmitted disease, there is very little risk of spreading HIV through normal heterosexual vaginal intercourse between two people not infected with another venereal disease. However, traumatic sexual practices, especially anal intercourse, involves significant risk of viral transfer by the blood that flows during these techniques.[8] In Africa, AIDS is primarily spread through heterosexual contact, but this is probably because of the significant incidence of venereal disease on that continent. It is postulated that alterations in genital mucosa associated with these diseases facilitate viral transfer through the now ineffective mucosal barrier. Ulcerative genital lesions are such a risk factor.[5b] Because HIV is primarily intracellular, with very little virus in body fluid, including plasma, the risk of contamination by noncellular fluid contact is remote.

Health-care personnel working with the AIDS patient are therefore at little risk for HIV infection unless they allow their skin barrier to be penetrated by the blood of an infected patient. This is most likely to occur with needle puncture; it may also happen if the skin barrier is broken, as by dermatitis.

Risk Groups

With the knowledge that blood is the most important vector of transmission, those groups most at risk for the disease may be identified. These include (1) male homosexuals, (2) intravenous drug abusers, (3) patients with hemophilia, (4) blood recipients, and (5) any sexual contacts of the aforementioned groups.

The risk to male homosexuals arises from their practice of anal intercourse and mixture of blood and semen. When anal intercourse is coupled with practices enhancing sexual gratification but at the same time causing trauma, especially bleeding in the rectal area, then the risk of viral spread is markedly increased. In 1989 the New York City Commissioner of Health estimated that 70% of the homosexual and bisexual population of that city is infected with HIV. Nationally, 67% of AIDS cases have occurred in these groups.

Intravenous drug abusers are becoming increasingly common as victims of AIDS and account for 23% of new cases, as contrasted with 57% of new cases in homosexual and bisexual men.[7] The communal use of contaminated needles and syringes by drug abusers, together with little regard for sterilization procedures, guarantees that any one infected individual will spread the virus to all other members of his or her group. It is estimated that 60% of drug abusers in New York City are HIV seropositive.

People with hemophilia and other recipients of blood products have been infected with HIV-contaminated blood administered before knowledge of the existence and dangers of AIDS and the development of effective screening procedures. One percent of AIDS in adults has occurred in hemophiliacs, whereas 5% of pediatric cases have occurred in hemophiliacs or children with other bleeding disorders who have received clotting factors.

Transfusion of contaminated blood or blood components has accounted for 2% of adult AIDS cases and 11% of pediatric cases.[7] Blood is now screened for presence of the HIV antibody, and positive units are not admitted to the donor pool. Although the number of new cases of HIV infection caused by the infusion of HIV-contaminated blood will decline, a significant number of blood recipients are already infected and will develop AIDS. Moreover, the fallibility of the ELISA screening will allow a few contaminated units to slip by, whereas the "window in time" of virus positivity, antibody negativity (as long as 14 months) introduces further risks. It is now estimated that the odds of contracting HIV infection by blood transfusion are 1 in 153,000 per unit transfused. A patient who receives the average transfusion of 5.4 units thus has odds of 1 in 28,000.[9] The risk has been decreasing more than 30% each year. Current donor-recruitment practices that seek to eliminate members of high-risk groups from the donor pool are estimated to eliminate 49 of every 50 donors likely to be HIV positive. Testing for the HIV antibody is 92% to 97% effective, and therefore the combined effectiveness of prescreening donors and antibody testing is 99.9%. Thus, blood and blood products are quite, but not absolutely, safe. The risk can be further reduced by transfusing fewer units, from fewer donors, recruiting more women and fewer men as donors, and encouraging more frequent donation from donors who have been tested repeatedly.

Clinical Syndromes

The patient acutely infected with HIV may remain completely asymptomatic for an indefinite period of time and may become aware of the infection only when a serious opportunistic infection develops. Acute infection may, however, be signaled by transient chills, fever, rash and malaise, which may occur at soon as 2 weeks after infection, but which usually disappear shortly thereafter.

Acute infection is followed by an indeterminate period of quiescence during which the virus invades and alters the lymphocytes, skin, central nervous system, peripheral nerves, and bowel. Contamination of bowel mucosa may result from direct introduction of the virus by anal sex or may simply reflect extensive spread of the virus into a wide variety of available and receptive cells. Profuse diarrhea is characteristic of AIDS and may be a direct result of HIV infection of bowel mucosa.[19]

Because there are so many and varied cell types subject to HIV infection, signs and symptoms are protean and confusing to the practitioner, and the disease may be difficult to diagnose until a major opportunistic infection announces its presence. The diagnosis is made more difficult because the practices that lead to its spread are unconventional and, in great part, socially unacceptable. It may be quite difficult to obtain a complete history from a patient unwilling to reveal unpleasant social practices.

What disrupts the harmony between the infective organism and host, and stirs quiescence into disease? Although the answer is not completely clear, there is evidence that the virus can mutate to pathogenic variants that push the patient from being in the carrier state to having AIDS. What causes the mutation is as yet unknown. There is good evidence that HIV infection always leads to AIDS and that AIDS always causes death.[21a]

Infection may first demonstrate itself as the AIDS-related complex (ARC). Diagnostic criteria have been created to distinguish ARC from AIDS, the major distinction being the absence of a life-threatening opportunistic infection or major tumor. This appears to be an arbitrary distinction. It is much more reasonable to think of HIV positivity, ARC, and AIDS as successive points on a curve of death.

Patients at risk for the development of AIDS include those who are positive for anti-HIV antibody and have (1) oral candidiasis, (2) intermittent or continuous fever for more than 1 month, (3) repeated night sweats, (4) debilitating fatigue, (5) persistent diarrhea, and (6) loss of body weight of 10% or more or who are anti-HIV positive and have failure of T cells to produce interferon gamma when stimulated by antigen.

The clinical manifestations of AIDS include (1) opportunistic infection, (2) Kaposi's sarcoma or other neoplasm, (3) anergy, (4) weight loss, (5) diarrhea, (6) adenopathy, (7) central nervous system dysfunction, (8) peripheral neuropathy, (9) renal failure, (10) cardiac arrhythmias or congestive heart failure, (11) arthralgia, (12) dementia, and (13) psychosis.

Opportunistic infections within the pulmonary system are important to the anesthesiologist because of the respiratory inadequacy and failure associated with them. *P. carinii* is a protozoan that is often resident in the lungs of healthy and asymptomatic people. Depression of the immune system allows overgrowth of the organism and development of a characteristic pneumonitis. In a patient with pneumocystis pneumonia the lungs grossly are rubbery, and the alveoli are clogged with a thick, gummy exudate, limiting gas exchange.

Tuberculosis is a major opportunistic infection in AIDS in Africa and is rapidly becoming common in the United States as well. AIDS must always be suspected in a young person with tuberculosis. It has been suggested that the tuberculosis of the AIDS patient may be particularly virulent and pose a risk to health-care workers caring for the patient.

Oral candidiasis is an important opportunistic infection of the esophagus, mouth, and pharynx. Erythema and edema are common and may be so extensive that they distort the anatomy of the upper airway and make endotracheal intubation extremely difficult.

Not all AIDS-associated pneumonias are caused by an opportunistic infection. Bronchiolitis obliterans organizing pneumonia has been described.[1] Much more common is nonspecific interstitial pneumonitis, which may be seen in as much as 32% of patients with AIDS.[27] These pneumonias probably are caused by the altered immune response and not by an opportunistic infection. Thus the diagnosis of pneumonia in an AIDS patient is complicated and may require the direct examination of tissue specimen to be definitive.

Cytomegalovirus may be isolated from more than 90% of patients with AIDS and, indeed, initially was thought to be the cause of the disease. This viral organism, too, is often found in asymptomatic, apparently healthy people, and immune suppression is probably necessary for its overgrowth. Cytomegalovirus may cause pneumonitis, which fortunately is rare but, unfortunately, is almost universally fatal. Hepatitis may be severe, and retinitis may cause blindness.

Other important opportunistic infections include cryptococcosis, toxoplasmosis, cryptosporidiosis, and herpes simplex. Many of these organisms are neurotoxic, creating confusing clinical pictures, and present the anesthesiologist with serious questions about the advisability of the use of regional anesthesia invading the neuraxis.

Kaposi's sarcoma is an angiosarcoma that classically, in the United States, has been described as a slowly growing ulcer of the lower extremity of elderly Italian or other Mediterranean men. The Kaposi's sarcoma of AIDS strikes young men, attacks lymph nodes and parenchymatous organs, is central rather than peripheral, and is found in the mouth and upper airway, where it may represent a hazard to endotracheal intubation.

Both Hodgkin's and non-Hodgkin's lymphoma are common in patients with AIDS. In New York City there has been a large increase in non-Hodgkin's lymphoma among the population at highest risk for HIV infection, i.e., men aged 25 to 54 years who have never married and who live in neighborhoods with high AIDS mortality.[14] Central nervous system lymphoma is common and can create a confusing clinical picture. Impingement of the airway by the lymphoma may make ventilation and intubation difficult, whereas intracranial lymphoma may cause serious problems with elevated intracranial pressure and its management during anesthetic administration.

Weight loss is caused by the profuse diarrhea so characteristic of AIDS. Painful candida infection of the mouth and esophagus make food ingestion difficult, and the patient often has anorexia and malaise, which further decrease food intake. Protein wasting will occur and cause hypoproteinemia. Electrolyte and fluid imbalance may follow extensive diarrhea.

Adenopathy is diffuse and extensive and is caused by reactive hyperplasia. It may be found wherever lymphoid tissue is found. Significant tonsillar and adenoidal hyperplasia have been described. Theoretically, lymphoid enlargement in the upper airway can constitute a hazard to ventilation and intubation.[2]

Anatomic pathologic and pathophysiologic conditions of the central and peripheral nervous system create serious problems for the diagnostician and for the anesthesiologist who may be called upon to deal with the afflicted patient. Vacuolar degeneration of the spinal cord occurs in 20% of AIDS patients and is characterized by paraparesis, ataxia, and incontinence. The clinical symptoms are weakness, unsteady gait, and loss of bowel and bladder control.

Subacute encephalitis is common in AIDS patients and causes progressive dementia. Clinically recognized dementia may occur in as many as 50% to 60% of patients with AIDS, whereas histologic changes in the cerebral cortex, found at postmortem examination, suggest that the total of both clinical and subclinical cases may be as high as 90% of all AIDS patients.

Facial paralysis may be the initial manifestation of AIDS. This otherwise rare clinical phenomenon, especially in a young person, should always cause the physician to consider HIV infection.

Peripheral neuropathies vary considerably both in severity and in clinical presentation. Diffuse, poorly localized pain, which may be mild to severe, is common, and analgesic therapy is needed in approximately 50% of hospitalized AIDS patients. Early symptoms and signs of peripheral involvement include diminished ankle reflexes and loss of appreciation of vibratory sensation.[26]

Differential diagnosis of central and peripheral nervous system dysfunction is complicated by the possibility of opportunistic infection within the nervous system. HIV has been isolated from cerebrospinal fluid, brain, spinal cord, and peripheral nerve of patients with AIDS-related neurologic complications, and invasion of nervous tissue occurs in most of the patients with neural abnormalities associated with AIDS or ARC. Demyelination and axonal degeneration within peripheral nerves are probably a direct result of HIV infection. In addition, HIV appears to cause vasculitis of the vasa nervorum of peripheral nerves, thus compromising the blood supply to nerves and, consequently, nerve function itself.

Renal failure is not uncommon in AIDS patients, and approximately 7% of those hospitalized with AIDS develop massive proteinuria, azotemia, or both (AIDS-associated nephropathy).[21] In most cases irreversible uremia develops. AIDS-associated nephropathy is characterized by proteinuria (>3.5 g urinary protein per day) and focal and segmental glomerulosclerosis. The prognosis is poor both in adults and children. Maintenance hemodialysis is not effective in prolonging life either in patients with AIDS-associated nephropathy and uremia, in patients with end-stage renal failure, or when AIDS develops during the course of maintenance dialysis.

Serious clinical cardiac abnormalities are common in patients with AIDS and are associated with myocarditis.[22] In one study postmortem examination revealed that 26 of 58 patients who died of AIDS had histopathologic myocarditis and 15 of 26 had clinical cardiac abnormalities, including congestive heart failure, ventricular tachycardia, electrocardiogram abnormalities, and pericardial abnormalities.[22] Although 55% of the patients did not have histopathologic changes, pericardial or electrocardiogram abnormalities were common. Overall, clinical cardiac abnormalities were found in 36% of the patients who died with AIDS.

Reiter's syndrome, reactive arthritis, polymyositis, and the sicca syndrome may herald the onset of clinically evident HIV infection and may also occur in patients with frank AIDS. HIV infection may mimic systemic lupus erythematosus.

In addition to dementia, psychosis may also occur in the AIDS patient, either as a consequence of the disease, or a psychotic predisposition that antedates the onset of AIDS. AIDS should be suspected in every patient presenting with psychosis of unknown origin and a history of homosexual practice or intravenous drug abuse.

PEDIATRIC AIDS

AIDS is not limited to adults, but the virus can be transmitted from infected mother to uninfected fetus in utero. By the end of 1989, 1995 children had developed AIDS, of whom 1080 (54.1%) had died.[7] The infusion of blood or blood products, either to treat hemorrhage or replace deficient clotting factors, played a role in transmitting the virus to children; however, this accounted for only 16% of the pediatric AIDS cases. The vast majority (81%) of children with AIDS are born either of mothers with frank AIDS or of those who belong to a high-risk group.

The incidence of in utero transmission of AIDS antibodies from mother to child is 100%. Thus, every child born of an anti-HIV-positive mother will also test positive for the presence of the antibody. Studies in Europe have demonstrated incidence rates of AIDS in babies born HIV-antibody positive to be 24%[19a] and 32.6%.[28] A New York study found that of those children who eventually developed AIDS, 20% developed the disease in the first year of life, and the remainder developed it at a nearly constant rate of 8% per year, reaching the median at 4.8 years.

THERAPY

Although researchers are trying to develop an AIDS vaccine, it is questionable whether an effective vaccine will ever be created, as alterations in the viral envelope present a constantly changing target for any vaccine. Even if one is developed, it is doubtful that it will be available in the near future.

The dependency of the virus on the enzyme reverse transcriptase is its ostensible Achilles' heel. The only effective drug that has had an extensive clinical trial is 3′-azido-3′deoxythymidine (AZT, zidovudine, azidothymidine), a reverse transcriptase inhibitor.[10] In the clinical trials the drug has decreased mortality and the frequency of opportunistic infections in patients with AIDS or ARC. Significant improvement has also been demonstrated in AIDS-associated neuropathy and in children with encephalopathy and dementia.

Unfortunately, while life may be prolonged and the quality of life improved with AZT treatment, there is no evidence that AZT will save lives. In addition, anemia and neutropenia are troublesome side effects of the drug. In one study of AZT in AIDS and ARC the major side effect was anemia: hemoglobin levels dropped below 7.5 g/dL in 24% of the patients receiving AZT but in only 4% of placebo recipients. Twenty-one percent of AZT recipients and 4% of placebo recipients required multiple transfusions. Neutropenia occurred in 16% of AZT patients and 2% of placebo patients.[23] AZT is beneficial but at a price that questions the cost versus benefit.

Dideoxycytidine is currently being studied. It appears that it may be combined with AZT in doses below the toxic level for each and produce an effective anti-HIV combination that has minimal toxicity.

A variety of agents have been found useful in treating the many viral, protozoan, bacterial, and fungal opportunistic infections of AIDS.[30] Acyclovir, vidarabine, and ganciclovir are effective antiviral agents, and isoniazid, rifampin, and ethambutol are useful in treating tuberculosis. Clotrimazole, amphotericin, and amikacin have found use in treating candidiasis. Unfortunately, resistance to acyclovir and ganciclovir has been reported.

P. carinii pneumonia has been treated successfully with pentamidine and cotrimoxazole. Both drugs, however, are toxic to most patients. Nevertheless, in an attempt to localize pentamidine to the site of infection and thus limit serious systemic effects, the drug has been successfully aerosolized for inhaled use.[18]

THE RISK TO HEALTH-CARE WORKERS

In 1983 the first case of a health-care worker who may have been contaminated with HIV from a patient and who subsequently developed AIDS was reported. A female surgeon from Denmark who was in none of the known risk groups developed profound wasting and diarrhea while working with the native population in Zaire. She apparently died of AIDS.[5a]

The Centers for Disease Control (CDC) reports that as of September 19, 1988, 169 health-care workers in the United States who had no evidence of high-risk behavior had developed AIDS ostensibly by contact necessitated by their professional activities.[6] At the time of the report 44 of the 169 had undergone extensive investigation, and no cause other than occupation could be ascribed as a factor in their illness. Of these 44, eight (18%) were physicians, including four surgeons.[6] It is thus clearly evident that the nature of their work clearly exposes health-care workers to a real risk of contamination with HIV and the development of AIDS.

The most significant risk is from puncture with a needle contaminated with HIV-positive blood. The estimated risk of viral transmission after puncture is 0.5% to 1.0%.[15] The risks of viral transmission and infection necessitate the incorporation of universal precautions, which are discussed later, in the elaboration of an anesthetic plan.

HAZARDS TO PATIENTS

The AIDS patient as a hazard to health-care workers has been emphasized; nonetheless, it is important to note that the health-care worker represents a danger to the immunosuppressed patient as well. Many people carry *Pneumocystis* within their lungs but do not become ill because their intact immune systems control the protozoan. Likewise, herpes simplex, which may be painful and unsightly, is self-controlled in those with intact immune systems. Each of these infections, however, may be devastating in the immunosuppressed individual. Precautions are necessary to protect medical personnel from the patient with AIDS; the same precautions help safeguard the patient from the medical personnel.

PREANESTHETIC ASSESSMENT OF THE PATIENT WITH AIDS

Because of the widespread and unrecognized prevalence of HIV infection, the practicing anesthesiologist will unknowingly deal with infected patients undergoing a wide variety of surgical procedures. Most patients have not been tested for the presence of the virus and thus their status is unknown. Testing for HIV without the patient's specific consent is illegal in most states. Health-care workers must incorporate the universal precautions, which will be discussed later, in their everyday patient management plans.

In the patient with AIDS, preoperative assessment demands evaluation of the presence and nature of any opportunistic infections and the use of appropriate therapy to control these infections. The pneumonias are especially important because of their ability to interfere with ventilation. Pneumocystis pneumonia presents special problems because of the thick, tenacious, gummy secretions that fill the alveoli.

Because renal dysfunction is quite common in the patient with AIDS, preoperative evaluation of renal function by determination of serum creatinine, blood urea nitrogen, and electrolyte levels is important. However, renal dysfunction in AIDS is progressive and has no known therapy.

Direct myocardial infection with HIV is common in AIDS. Chest radiographs and electrocardiograms should be routine assessment tools. If there is evidence of congestive heart failure, determination of cardiac output and cardiac ejection fraction may be indicated. A decrease in blood volume caused by diuretic therapy may make the cardiac pump more efficient but whether the infected heart will respond to inotropic agents is unknown.

Prolonged and excessive diarrhea may produce dehydration and electrolyte imbalance. Oral repletion may be difficult because of the frequently occurring and extensive candida infection of the mouth and esophagus. Adequate rehydration and correction of electrolyte abnormalities are necessary before extensive anesthesia and surgery and may require total parenteral nutrition to correct the state of malnutrition.

Hepatic dysfunction is common in AIDS patients because of concurrent infection such as hepatitis B or an opportunistic infection (e.g., hepatitis induced by cytomegalovirus). Liver function tests should be routinely performed in AIDS patients.

Coagulopathies, usually platelet dysfunction or thrombocytopenia, occur in the AIDs patient. This decrease in platelet number and activity may result from viral suppression of bone marrow function or AZT therapy. If the patient has a history suggestive of bleeding dyscrasia (easy bruising, petechiae, and so forth) a template bleeding time should be performed and platelet infusion considered to correct a prolonged bleeding time. Extensive liver dysfunction may contribute to a bleeding diathesis by inhibition of formation of individual clotting factors.

The airway must be evaluated carefully. Hypertrophy of tonsils and adenoids may cause airway obstruction. Kaposi's sarcoma may occur in the upper airway and make intubation difficult. Oral candidiasis is a common opportunistic infection in AIDS patients and can cause significant edema and erythema that is sufficient to alter the anatomy of the upper airway to the extent that endotracheal intubation using conventional techniques may be difficult to impossible. In a patient with extensive oral candida infection, awake visualization of the airway should be performed and ease of intubation assessed with the patient breathing spontaneously. If the anatomy is distorted by the infection, respiratory depressants and muscle relaxants must not be used until the airway is secured either by awake intubation or tracheostomy. Guided intubation by fiberoptic laryngoscopy or bronchoscopy may be impossible because of severe anatomic deformity, erythema, and edema.

ANESTHETIC MANAGEMENT

The choice of anesthetic agent and technique must be individualized to the patient's state of health and surgical requirements. Because of their negative inotropic properties inhalation anesthetic agents should be used with great caution (if at all) in the patient with a myocardium compromised by viral infection. Intravenous narcotics, with no effect on inotropy, may be the drugs of choice. Major conduction anesthesia, especially those techniques invading the neuraxis may be contraindicated because of viral infection of the central and peripheral nervous systems and the possibility of further dissemination of the infection. This dissemination of an infective agent has not been demonstrated and may be of more medicolegal than medical concern. However, AIDS is progressive, and neuropathies are common. Regional anesthesia should be used if it is clearly the technique of choice and the patient has been adequately advised of the risks of the procedure and the probable increase in neurologic signs and symptoms.

MONITORS

Monitoring the blood pressure, pulse, and electrocardiogram in the AIDS patient is as important as in the patient who is not HIV infected. Because adequate ventilation of the patient with pneumocystis pneumonia or diffuse tuberculosis may be difficult, pulse oximetry and capnography will be of special importance. The high incidence of virus-induced cardiac disease should broaden the indications for the use of invasive monitors, especially the pulmonary artery catheter. It has been suggested, however, that invasive monitoring may be hazardous because of sympathetic dysfunction and hypotension.

UNIVERSAL PRECAUTIONS

The CDC recommends that all patients be considered as potential hazards to health-care workers and that the following universal precautions be used with all patients.

1. Barrier precautions should be used when contact with blood or body fluid of any patient is anticipated.
2. Gloves should be worn when handling body fluids, mucous membranes or nonintact skin. There are no reported differences in barrier effectiveness between intact latex and intact vinyl used to manufacture gloves. Thus, the type of gloves selected should be appropriate for the task being performed.
3. Mask and protective eye-wear should be worn when droplets are formed.
4. Handwashing is very important, and should be done frequently, and especially after patient contact.
5. Needles should be handled with great care and never recapped.
6. Mouth-to-mouth resuscitation should be minimized, and adequate ventilatory equipment should always be available.

7. Health-care workers with dermatitis should not be involved in direct patient care.
8. Pregnancy involves no special risk, but the pregnant health-care worker should exercise all precautions.

These precautions are applicable not just to AIDS, but to other infectious diseases, especially hepatitis.

SUMMARY

HIV infection is widely prevalent and the infected patient may present with varied and protean signs and symptoms. Opportunistic infections of the airway, lungs, and central and peripheral nervous systems challenge the anesthesiologist to develop an effective management plan. The patient's condition must be optimized before induction of anesthesia and surgery by appropriate treatment of the infection and evaluation of possible barriers to ventilation and intubation. No anesthetic plan is ideal, and there may be only limited indications for regional anesthesia.

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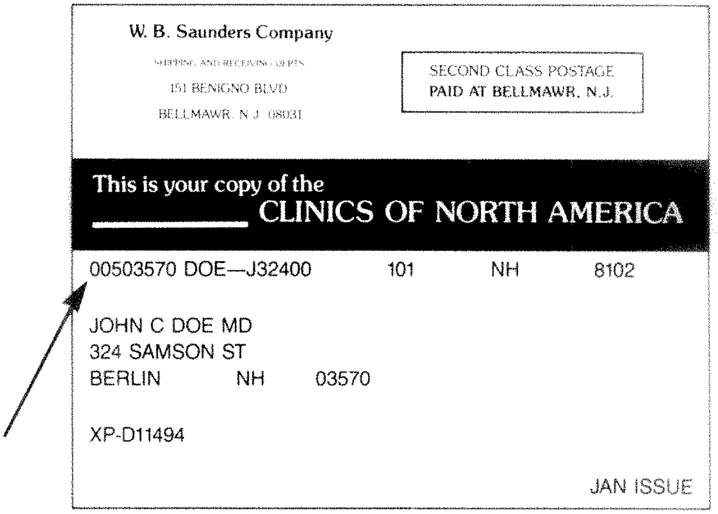

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